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Doppler Indices of Gas Phase Formation in Hypobaric Environments: Time-Intensity Analysis

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SUMMARY

1. A semiquantitative method to analyze decompression data is described; it possesses the advantage that it allows a **graded response** to decompression rather than the **dichotomous response** generally employed.
2. A generalized critical volume (C-V) or stoichiometric (time-independent, equilibrium) model is examined that relates the constant of the equation $P_i = mP_f + b$ to variable tissue supersaturation and gas washout terms. This equation has classically been used to describe ascent limiting conditions (*M*-values).
3. The effect of the tissue ratio (TR) on gas phase formation indicates that a decreased ratio yields fewer individuals with Doppler-detectable gas bubbles, but those individuals still present with Spencer Grade III or IV. This might indicate a local collapse of tissue supersaturation.
4. The individuals with Spencer Grade III or IV could be at risk for Type II decompression sickness by transpulmonic arterialization.
5. The primary regulator of the problems of decompression sickness is the reduction of local supersaturation, presumably governed by the presence and number of gas micronuclei. It is postulated that a reduction in these nuclei will favor a low incidence of decompression sickness in microgravity secondary to hypokinesia and adynamia.

For the last century, the data derived from studies of decompression sickness have been treated as dichotomous, that is, "yes" or "no" results; fine points and details are lost in this method of analysis. While it is known that joint-pain decompression sickness is not an all-or-none phenomenon, a **dichotomous treatment** of the data has shaped our thinking. Artifices such as "subclinical decompression sickness," "niggles," and "silent bubbles" are engendered to connote a **continuum** which is suspected to exist.

Here is described a **numerical relationship** between the Doppler-detectable gas phase and the variations in the occurrence of decompression sickness. It is one attempt to render Doppler data into other than an ordinal form; other systems exist (Eatcock and Nishi, 1987). It must ever be recalled, however, that the major portion of the gas phase detected is almost assuredly not spawned in the tendons and ligamental connective tissue responsible for decompression sickness.

I. DECOMPRESSION ACROSS THE HYPO/HYPERBARIC CONTINUUM

A. Gas Phase With Inert Gas Alone

It is not obvious that there exists a continuum of the decompression spectrum as one moves from hyperbaric to hypobaric conditions. In some cases, it can seem as though hypobaric decompressions are greater in the pressure "jumps" that can be sustained without injury. It would be desirable to be able to apply some of the data from deep-sea diving (especially saturation diving) to the question of hypobaric decompression.

The question of the linearity of decompression data across the pressure continuum has been addressed in the past (Hills, 1966; Hennessy and Hempleman, 1977), and it will be treated here, using analysis similar to the quoted works but indicating my reasoning when points differ (primarily with the interpretation of the various constants and their application to hypobaric cases).

From an analysis of decompressions made from saturated conditions, Hennessy and Hempleman showed that decompressions over a large pressure continuum could be represented by the formula

$$P_i = \alpha P_f + b \quad (1)$$

where

P_i = initial saturation pressure

P_f = final (lower) pressure of decompression stage to produce decompression sickness

α = slope

b = intercept

For the case of decompression from normoxic helium,

$$\alpha = 1.397 \text{ and } b = 0.57 \text{ [atm]}$$

for normoxic air,

$$\alpha = 1.361 \text{ and } b = 0.34$$

This is depicted graphically in figure 1.

Probably the greatest amount of data concerning decompressions in the hyper- and hypobaric continuums comes from Professor Bühlmann (1984); this results from the particular interest of diving in Switzerland where pressures vary above 1.0 atm. Bühlmann's analysis of decompressions from 0.47 to 1.5 atm indicates that it is useful to consider a tissue with half times as long as 600 minutes. If this is done, the Swiss data can be displayed in the linear fashion according to the equation

$$P_i = 1.0395 P_f + 0.245 \quad (2)$$

A safe decompression from 1 atmosphere ($P_i = 0.79$ ata) to 0.51 ata for a TR of 1.54 could be performed according to this equation. The maximum TRs of the longer half-time tissues were **constrained**, however, to **restrict ascent** from sea level to an altitude no higher than 18,000 feet.

Concerning the question of the proper over-pressure for the slow tissue, Bühlmann writes:

During the last 20 years, few experiments with saturation with air have been performed in comparison with exposures with saturation with helium. In the experiments, pains in the joints occurred very frequently during decompression, an important argument for the belief that the tolerance of the slow tissues was less than had been assumed for a long time.

In the discussions of both Hills (1966) and Hennessy and Hempleman, the values of the constants a and b are given as solubilities, liberated gas volumes, and additional tissue pressures consisting of both surface tension and tissue deformation pressures. **Essentially all of the dissolved gas is expected to come out of solution during decompression;** the presence or absence of symptoms depends only on the released volume.

A treatment by Yount (1979) relates the values to the permanent presence of gas micronuclei that can expand during certain specified pressure profiles. The relatively good agreement between the theory and the data of Berghage indicates a problem since Berghage's study did not deal with the "bends" but rather with a complex process of pulmonary gas embolism and death. My analysis indicates that the values generated are not generally compatible with the biological situations. That is, either unreasonable values must be assumed for the surface tension and deformation pressure, or proposed decompression-generated tissue gas volumes are too large.

Both Hills (1966) and Hennessy and Hempleman found that

$$a = (1 + V_c/\beta), \text{ and} \quad (3)$$

$$b = a(2\gamma/r + \delta) \quad (4)$$

where,

V_c = critical volume, i.e., that just sufficient to produce limb-bend decompression sickness

β = solubility of the inert gas in the tissue producing the limb-bend pain (probably tendon and/or ligament)

r = radius of the tissue gas bubble responsible for the pain

γ = surface tension

δ = tissue deformation pressure (Nims, 1951)

The analysis in the appendix gives a somewhat different interpretation to these values. It is similarly derived from the stoichiometric analysis of gas content in a simple physical system, is more general and more applicable to hypobaric conditions, and also accords more accurately with some aspects of the pathophysiology of decompression sickness. It is a static method of analysis that does not allow for the generation of time-dependent phenomena (see, for example, Liou and Wissler, 1987)

but is a useful scheme for the conceptualization of decompression data. In this case, we find that for the M -value relationship

$$\begin{aligned}
 P_i &= mP_f + b \\
 m &= \frac{[V/\beta]}{[(1-\sigma)(1-\lambda)] + 1} \\
 b &= \frac{\delta[V/\beta]}{[1 - (\sigma)(1 - \lambda)] + 1}
 \end{aligned} \tag{5}$$

where σ is that fraction of the system that remains supersaturated following a decompression, λ is a loss factor, and δ is the internal pressure; for example, surface tension or the tissue deformation pressure of Nims.

This concept of supersaturation is meant to reside in a volume of tissue and not to represent a microregion next to a gas bubble itself. Here, of course, the value of σ is approximately zero. In general, most of the inert gas taken up by tissues remains in the supersaturated condition during decompression (Powell, 1972b; Powell, 1973; Powell and Weydig, 1974; Hills, 1978; Powell, Spencer, and von Ramm, 1982). The idea of some allowable tissue gas phase formation is seen in the treatment of Liou and Wissler.

The constant λ is a loss factor which derives from the fact that dissolved inert gas is lost from the tissues by virtue of the blood flow during the decompression (reduced pressure) phase. A reduction of σ with a reduction in λ (perfusion) will result in the development, in time, of decompression sickness (if V_c is reached).

Because of the value of the non-zero intercept in equation 1, the pressure change ratio for not incurring decompression sickness is greater in hypobaric cases. This gives the appearance that other processes are playing a role in altitude decompression sickness when compared to the problems of deep-sea divers. This may not necessarily be the case.

It has been noted that the direct application of the M -value concept of Workman cannot be used to extrapolate suitable flying-after-diving values (Nishi, 1989). This results from the non-zero intercept of the relationship of M -value vs. the ratio of initial and final pressure. To correct the M -values, both Bassett (1982) and Nishi used a ratio

$$Ro = \frac{Mo}{[\text{ambient pressure}]}$$

Nishi described the ratio as

$$R = a\{1 + [b/(D + P_{\text{surface}})]\} \tag{6}$$

where

$$a = M_{10} - M_0 = M \text{ and,}$$

$$b = [M_0/M] - P_{\text{surface}}.$$

While useful, the numbers derived have not been tested; the system is an extrapolation. Since much of altitude decompression work involves breathing pure oxygen and exposure to pressures

relatively near to the partial pressures of oxygen, water vapor, and carbon dioxide, these topics will be discussed in more detail in later sections.

B. Gas Phase Including Non-Inert Gases

For purposes of this discussion, we might refer to the gases not necessarily present in tissues or entering into biochemical reactions (e.g., nitrogen and helium) as "xenogenous gases" (Greek ζ ενοο, "stranger") or "exogenous" (Greek εξοο, "without" or "outside") to distinguish them from gases normally present in the body and entering into biochemical reactions, viz. oxygen, carbon dioxide, and water vapor. The tissue partial pressures of these endobiotic (Greek ενδοο, "within") gases are approximately:

oxygen	= 40 [torr]
water	= 47 [torr]
carbon dioxide	= 40 [torr]

for a total of 127 [torr]. At depths, these partial pressures will be insignificant; and even at sea level, they are but a modest fraction of the total of all partial pressures comprising the gas phase, viz. $130/760 = 17\%$. As the ambient pressure decreases, they assume a greater importance since they will enter a gas bubble by diffusion (and probably enter into its formation). The entrance, or exit, is given by Van Liew (1968):

$$dV_i/dt = (kA/I)(P_i - P_o) \quad (7)$$

where

- V_i = volume of the i th gaseous component
- k = is related to the diffusivity and solubility in the boundary layer
- A = surface area of the bubble
- I = length of the diffusion boundary
- P = partial pressure of the component inside and outside of the bubble

The percent partial pressures of endobiotic gases for different altitudes are:

Sea level	= 17%
20,000 ft	= 37%
30,000 ft	= 58%
40,000 ft	= 92%

The relative contribution of these to clinical decompression sickness is not altogether clear. The numbers imply that an increasingly larger role is played by the endobiotic gases as altitude increases or, conversely, that there will exist a maximum in altitude that is time invariant to which one can safely decompress.

II. CLASSICAL DOPPLER ANALYSIS OF DECOMPRESSION DATA

It has been customary in many laboratories to analyze Doppler data on the basis of bubbles detected (+) or bubbles not detected (-). Such a method is operationally easy but wastes a considerable amount of the information in the Doppler data. Under this system, a subject with a few gas bubbles detected only once in the course of a 3-hour monitoring session would count as a "+" with the same weight in the analysis as a subject with Spencer Doppler Grades of III and IV detected for 2 hours.

An analysis of hypobaric decompression data in accordance with this system is shown in figure 2. Here we see data from Conkin et al., (1987) in which the number of subjects with any Doppler-detectable bubbles is plotted vs. the TR in the 360-minute tissue compartment as determined by calculation. The line will eventually intersect the x-axis since the number of individuals with any detectable gas bubbles will eventually become zero (or at least the number and radius of the bubbles will eventually make their detection impossible). This analysis will be compared with that derived from time-intensity analysis methods to be discussed in section III.

III. RATIONALE FOR TIME-INTENSITY ANALYSIS IN HYPOBARIC ENVIRONMENTS

The first successful formulation of decompression schedules was devised by J.S. Haldane (Boycott et al., 1908) using the concepts of multiple tissues and exponential uptake and elimination of inert gas resulting in tissue half times. Analysis of the concept and the testing of tables was for many years by means of "bends/no bends" endpoints. Newer diagnostic technologies, in the form of ultrasound, have arisen since the late 1960's.

Ultrasound has been employed in the modalities of through-transmission, two-dimensional imaging, and Doppler-shifted flowmeters. The ultrasound technique has been used for approximately two decades to evaluate decompression tables for deep-sea divers. In the early 1970's, this was done by simply determining the presence or absence of precordial Doppler-detectable gas bubbles. Powell (1974) introduced the concept of "probability of decompression outcome" associated with the maximum precordial Doppler grade in a study in which pigs were the subjects.

In 1975, this probability concept was expanded to include men (Powell and Johanson, 1978); a parallel study by Nashimoto and Gotoh (1978) reached similar conclusions. Gardette (1979) extended the study to include saturation decompressions. In recent years, the technique has been expanded to include many subjects in saturation, "bounce" and altitude exposures; a compilation by Vann et al. (1982) includes more than 1000 individuals and relates their Spencer Doppler Grade (Spencer and Johanson, 1974) to the final outcome of their decompression. The result is shown in table I.

Table I
Relationship of Precordially Doppler-Detectable Gas Bubbles and
Decompression Sickness

	Maximum Spencer Doppler Grade:				
	<u>0</u>	<u>I</u>	<u>II</u>	<u>III</u>	<u>IV</u>
(1) Saturation	0/33*	0/4	3/11	2/2	2/2
(2) Sub-saturation	2/471	1/140	1/84	13/103	17/52
(3) Altitude	0/75	1/6	2/12	8/25	29/55

*[Number of subjects with DCS] / [total subjects with grade]

As an extension of the method that uses the maximum Spencer Doppler Grade determined for each decompression, a "time-intensity integral" method to determine decompression stress has been employed by Nishi et al. (1980) and Powell (Powell and Spencer, 1981). This consists of an integration

over time of the precordially determined Spencer Doppler Grade. The decompression stress (Ψ) is then,

$$\Psi = \int V dt$$

Nishi's formula was

$$\Psi = \{100/4^a (t_j - t_o)\} \sum_{i=1}^j [t_i - t_{i-1}] (d_i^a + d_{i-1}^a)/2 \quad (8)$$

where

d = Spencer bubble grade at t_i , ($d_o = 0$)

j = number of observations

t_i = time of last observation ($t_o = 0$) land

a = a parameter to adjust for the nonlinearity in the Doppler bubble grade

An alternative method based on elevations in the right ventricular systolic pressure with gas embolization (*vide infra*) was used to adjust for nonlinearity in the Spencer Grades of Doppler data. That method and its results form the basis for this report.

The time-intensity index was originally proposed to evaluate Doppler ultrasound data from decompression profiles of differing stresses; because the index shares problems inherent in all forms of Doppler bubble detection, it was less than optimally successful. (The problems alluded to refer to the fact that the Doppler gas phase is not spawned exclusively in the tissue responsible for joint pain but rather primarily in muscle tissue.) It will be used in this presentation as an alternative to evaluating decompression data as "bubbles/no bubbles" in an investigation of the effects of the TR on the outcome of a simulated flight.

The decompression stress index concept can trace its origins to through-transmission ultrasound studies performed on rats exposed to differing decompression stresses (Powell, 1972). The degree of gas phase formation (time-intensity) in thigh tissue parallels these stresses. For the gas phase determined in the precordial mode, varying decompression stresses (achieved through a variation in the bottom time) yielded a regular and orderly progression in both the intensity and duration realm (Powell and Spencer, 1980; figure 3a). Another example could be found in the analysis of Doppler data from subjects exposed at the same pressure for the same duration but under differing environmental conditions (e.g., warm/wet, cold/wet, warm/dry, etc.) (Powell and Spencer, 1981; figure 3b).

Time-intensity analysis was used by Dunford and Hayward (1981) and Eckenhoff et al. (1989) in the analysis of their diving data; in the case of the latter, it was used for (hyperbaric) decompressions from saturated conditions.

Since the concept of decompression stress is independent of the initial starting pressure (one atmosphere absolute is a valuable reference point for work on this planet's surface, but it holds no particular physical significance), the time-intensity technique can be applied equally well to decompressions to altitude.

The data from precordial Doppler ultrasound monitoring is currently depicted in a form that relates a given sound pattern to a number referred to as the "grade." The most commonly used method is the one first published by Spencer and Johanson (1974); it is, however, only a relative scale and does not directly relate to a given quantity of gas. One method to convert from the Spencer Grade to a gas

volume is the quantitative measurements of Powell and Spencer (1980) which relate the volume of the gas phase present in the pulmonary artery with the Doppler grade, both having been simultaneously measured. To determine the volumes, two steps were required. The first step related the rise in right ventricular systolic pressure (RVSP) to the volume of air introduced during air injection through a catheter. The second step required that the rise in RVSP be measured, after decompression, while the Spencer precordial Doppler Grade was measured.

These gas volumes were determined at steady state and, as such, are possibly different from the short "bursts" heard in the hypobaric chamber following limb flexure. Nonetheless they represent a method of conversion which has considerable physiological basis. The relationship of RVSP and the volume of gas injected into the pulmonary artery is shown in figure 4; the volumes and Spencer Doppler Grades are indicated in table II.

Table II
Vascular "Gas Volume" as a Function of Spencer Doppler Grade

<u>Doppler Grade</u>	<u>Volume [cc/kg-min] $\times 10^2$</u>	<u>Relative Volume</u>
0	0	0
I	<1	1
II	1.0	4
III	1.6	6
IV	2.5	10*
IV+	3.5	10*

*IV and IV + were equated as equal for purposes of this analysis.

Doppler grades as a function of time were tabulated from data of hypobaric decompression experiments determined earlier at NASA/JSC and described in Conkin's study. These will produce curves of the type shown in figure 5 where the Doppler gas volume for six different individuals is plotted as a function of time. The curves appear similar to those in figure 2. In this type analysis, a continuous grade of IV would bear a considerably greater numerical weight than an intermittent grade I; if simple "yes" or "no" results were sought with respect to bubble presence, both would count equally. All subjects averaged together produce curves of the type shown in figure 6.

A measure of decompression stress can thus be calculated by integrating the area beneath the curve of Doppler Gas Volume *vs.* Time (in minutes) for all of the subjects exposed to a decompression which resulted in a given TR. This TR is the ratio of inert gas (nitrogen) in a tissue (taken in these cases to be the 360-minute half-time tissue; see Conkin's study) to the ambient (hypobaric chamber) pressure.

The decompression stress ($\Psi = \int V dt$) for various values of TR (360 min) in conjunction with their respective standard errors of the mean (SEM) is given in table III. While the average Doppler gas volume is larger as the TR increases, the variation among the tests can be considerable and SEMs can overlap in varying degrees.

Table III

**Decompression Stress as a Function of Tissue Ratio Calculated for the
360-Minute Half-Time Tissue**

<u>Tissue Ratio</u>	<u>Decompression Stress</u>	<u>Std. Error of Mean</u>
1.83	8.62	2.25
1.80	5.36	2.14
1.75	4.11	1.80
1.70	2.21	0.68
1.68	1.62	0.44
1.67	2.89	1.61
1.60	3.76	0.95
1.45	0.47	0.29
1.43	0.34	0.14
1.35	1.40	--
1.35	1.54	1.07
1.32	0.55	0.54
1.38	0.09	0.07
1.12	0.38	0.36

Some decompressions are repetitive.

IV. DEVELOPMENT OF AN IN VIVO GAS PHASE

A difference could not be found between the Doppler "gas volume" (decompression stress) induced by decompression to a given TR whether it had been produced by oxygen prebreathing followed by a large decompression step or by direct decompression with a smaller step and without prebreathing.

The tissue half-time concept introduced by Haldane (Boycott et al., 1908) was considered to represent a condition of supersaturation which was time-independent. In recent times, the concept has been advanced that the "faster" tissues will support a higher degree of supersaturation because they need sustain it for only a relatively short period of time; that is, the dissolved inert gas is rapidly washed out. The "slower" processes (or compartments or tissues) are of greater interest to hypobaric physiology since it is the dissolved inert gas in these slower processes that limits the degree of decompression from sea level pressure to altitude.

Decompression to altitude, with return to a higher pressure after a period of time, differs from decompression to sea level (as would be performed by a deep-sea diver) in that the latter is **unbounded in time for the development of decompression sickness**. An aviator or astronaut can perform a decompression (or upward excursion) that can be severe with regard to the TR and probably not incur decompression sickness if the length of time spent at the lower pressure is restricted. In terms of the model presented here, this safe period exists because the reduction, or breakdown, of supersaturation is time-dependent.

If blood were to cease flowing before the instigation of decompression, the *in vivo* situation would be represented by equation 10A and the family of curves shown in figure A-2. This illustrates the situation of a closed system which is decompressed with the fluid contained within a condition of saturation. With the passage of time, the degree of supersaturation will decrease and a given volume of gas will separate out. This will not only change the value of σ , but it will also modify the factor identified as "loss" since, in this context, the gas-saturated fluid is lost through blood perfusion.

The ratio P_i/P_f is then independent of time for the case of the deep-sea diver, but it is not independent of time for the astronaut. The initial TR that can be modified by oxygen breathing will influence the average time to the appearance of problems of decompression sickness (and of the appearance of Doppler-detectable gas bubbles) (Waligora et al., 1987). At a given ratio, the degree of gas phase formation (as measured by the Doppler flowmeter) can be increased by muscle activity (which functions to reduce the supersaturation) (Krutz and Dixon, 1987).

From the aspect of deep-diving data and an analysis of Conkin's Doppler data, an acceptable TR to use during ascent can be thus described

- For the time-independent case, it could be stated that the value developed from deep diving (Bühlmann, 1984), $TR = 1.54$, is too large when calculated on the basis of the 360-minute half-time tissue.
- For times longer than 2 hours at reduced pressure, the acceptable TR cannot be greater than 1.1 if one wishes to carry a risk of decompression sickness. This may be of only the mildest form however.

Because of the possibility of transpulmonic arterialization, Type II decompression sickness is not totally avoidable. It is necessary to determine those physiologic situations in which an embolic cerebral gas phase might be present.

V. TIME-INTENSITY ANALYSIS AND CLASSICAL DOPPLER ANALYSIS

A. Correlation to the Tissue Ratio

In Conkin's study, a classical analysis was made in which the number of subjects with Doppler-detectable gas bubbles was compared to the TR; the best correlation appeared when the 360-minute tissue half time was used (figure A-5).

A similar analysis of correlation was made for the time-intensity method. Tissue inert gas loadings were calculated for all simulated ascents described by Conkin's study for which "gas volumes" could be calculated; these are given in table IV. Figures 7 through 9 show the "fit" of a plot of the gas volume *vs.* the 240-, 360-, and 540-minute tissue half time, respectively. A combination of the TRs for both the 360- plus the 540-minute half time appeared best for Doppler "gas volume" (figure 10).

Table IV

Calculated Tissue Inert Gas Tensions

1st line = tissue inert gas tension (in fsw)

2nd line = tissue ratio

<u>90 min</u>	<u>120 min</u>	<u>240 min</u>	<u>360 min</u>	<u>420 min</u>	<u>540 min</u>
<u>Profile 1 (in Conkin et al., 1987):</u>					
5.17	7.75	14.21	17.40	18.43	19.91
0.54	0.80	1.47	1.80	1.91	2.06
<u>Profile 2</u>					
17.05	17.16	18.11	19.20	19.66	20.48
1.77	1.78	1.88	19.9	2.04	2.12
<u>Profile 3</u>					
8.53	10.20	13.99	16.21	17.04	18.35
0.88	1.06	1.45	1.68	1.77	1.90
<u>Profile 4</u>					
3.80	6.14	12.65	16.09	17.24	18.89
0.39	0.64	1.31	1.67	1.79	1.96
<u>Profile 5</u>					
10.34	11.79	15.04	17.01	17.76	18.95
1.07	1.22	1.56	1.76	1.84	1.96
<u>Profile 6</u>					
3.26	5.47	11.94	15.48	16.68	18.41
0.34	0.57	1.24	1.60	1.73	1.91
<u>Profile 7</u>					
3.22	5.40	11.28	13.80	14.52	15.50
0.33	0.56	1.17	1.43	1.50	1.61
<u>Profile 8</u>					
10.32	11.71	14.61	16.38	17.09	18.25
1.07	1.21	1.51	1.70	1.77	1.89
<u>Profile 9</u>					
10.31	11.66	13.59	13.92	14.02	14.28
1.07	1.21	1.41	1.44	1.45	1.48

Table IV - concluded

<u>90 min</u>	<u>120 min</u>	<u>240 min</u>	<u>360 min</u>	<u>420 min</u>	<u>540 min</u>
<u>Profile 10</u>					
10.32	11.71	14.61	16.38	17.09	18.25
1.07	1.21	1.51	1.70	1.77	1.89
<u>Profile 11</u>					
5.59	6.12	8.63	10.91	11.88	13.55
0.58	0.63	0.89	1.13	1.23	1.40
<u>Profile 12</u>					
10.3	11.61	13.24	13.39	13.45	13.70
1.07	1.20	1.37	1.39	1.39	1.42
<u>Profile 13</u>					
5.59	6.10	8.09	9.30	9.76	10.55
0.58	0.63	0.84	0.96	1.01	1.09
<u>Profile 14</u>					
10.30	11.61	13.21	13.19	13.09	12.95
1.07	1.20	1.37	1.37	1.36	1.34
<u>Profile 15</u>					
5.59	6.10	8.08	9.20	9.55	10.06
0.58	0.63	0.84	0.95	0.99	1.04
<u>Profile 23</u>					
1.76	3.26	9.21	13.02	14.38	16.40
0.17	0.34	0.95	1.35	1.49	1.70
<u>Profile 24</u>					
0.65	1.63	6.51	10.33	11.79	14.06
0.07	0.17	0.67	1.07	1.22	1.46
<u>Profile 25</u>					
17.86	18.10	18.47	18.71	18.87	19.23
			1.37	1.39	1.40
<u>Profile: Exercise Study of K.V. Kumar, MD</u>					
25.84	25.89	25.96	25.99	26.00	26.01
1.77	1.77	1.78	1.78	1.78	1.78

The calculation of decompression stress was originally undertaken to allow comparison between different decompression profiles; however, the large SEMs often precluded making a comparison that was statistically valid.

In preparing graphs in the form of log-log plots, we assume that a critical degree of supersaturation (cf. Boycott, Damant, and Haldane, 1908) does not indeed exist; some tissue gas should be evolved with any degree of supersaturation. There will, no doubt, be an indistinct lower boundary where the minimally evolved gas will be clinically insignificant and/or short lived and virtually undetectable.

The Doppler gas volume, V_g , and the supersaturation (= tissue ratio, TR) can then be described by

$$V_g = a [TR]^b \quad (9)$$

or

$$\log V_g = \log a + b \log [TR] \quad (10)$$

Equation 9 is similar in form to the original square root law of Hempleman (Hempleman, 1982) and the isopleths of Doppler-detectable venous gas bubbles as described by Spencer (1976).

B. Analysis of Repetitive Ascents

If one examines the results of decompressions by means of classical Doppler analysis (i.e., bubbles/no bubbles), the scatter of data leads one to believe that there is no difference between single excursions and ones made somewhat later on the same day. The second excursion of the day is depicted by unfilled diamonds (graphically illustrated in figure 11).

When a similar analysis is performed with the gas volume method, all of the second (i.e., repetitive) decompressions (depicted by the solid triangles) are clearly above the line in figure 12. This indicates that the volume of gas generated upon decompression for the second time is greater than that expected for a single (i.e., first) decompression on that day.

C. Collapse, or Breakdown, of Tissue Supersaturation

Not all subjects develop a measurable venous gas phase as determined by the Doppler flowmeter, and there is a considerable degree of apparent randomness introduced into the data. This results in a large SEM. Comparisons of Doppler results analyzed with either the classical or the gas volume approach yield similar answers since a large fraction of those subjects which develop a measurable vena caval gas phase present with either Grade III or IV (see table V).

This high percentage of subjects with Grade III or IV is postulated to be the result of the breakdown of supersaturation in the tissues with the generation of a gaseous phase which could become considerable. This was noted to occur in rats decompressed from gradually lengthened bottom times (Powell, 1971). The appearance of a large *in vivo* gas phase was quite sudden, it being impossible to gradually titrate the rats from mild to severe decompression sickness symptoms.

In terms of equation 5-A (figure A-2) and for purposes of illustration, it is possible to give a quantitative estimate of gas phase evolution. A system ($P_i/P_f = 4$) producing about 1/2 cm³ of a (tolerable) gas phase because of a high degree of supersaturation (90%) would suddenly produce approximately 4 cm³ if the supersaturation fell to 0%.

Table V

**The Decompression Tissue Ratio and the Number of Subjects
Presenting with Spencer Grade III or IV Doppler-Detectable
Precordial Gas Bubbles**

<u>Tissue Ratio</u>	<u>% Subjects with Bubbles</u>	<u>With Grade III to IV</u>
1.83	89	12/14
1.80	64	5/7
1.75	45	9/10
1.70	65	11/12
1.68	57	15/26
1.67	66	6/7
1.60	46	12/13
1.45	36	1/4
1.43	42	3/4
1.37	33	3/4
1.35	33	3/4
1.32	29	3/4
1.24	10	1/2
1.12	17	1/2

The effect at the local tissue level would additionally be to reduce the local perfusion and therefore modify the loss factor which reduces the number of moles of dissolved tissue gas with time. In terms of equation 8-A (figure A-3), a system with a high loss of tissue gas in the dissolved state (by means of blood flow) could find that loss is reduced secondary to capillary blockage. The combined effects are evident in figure A-4 which illustrates the degree of decompression that a tissue system can tolerate without incurring clinical evidence of decompression sickness (either Type I or II). This figure is generally employed to graphically illustrate the ascent criteria in the *M*-value method of table calculation.

If, as appears to be the general case, some individuals can develop a large tissue gas phase (as identified by presenting with Grade III or IV Doppler-detectable precordial bubbles), it might be expected that there may indeed not exist a lower boundary for the development of decompression sickness. This lower boundary was a cornerstone of the original treatment of Damant, Boycott, and Haldane. However, a reasonable fit of gas phase evolution *vs.* tissue ratio can be found in equations of the log-log type as was shown earlier.

Naturally, the occurrence of decompression sickness can be related to the decompression stress (the Doppler "gas volume") since the latter is of the data set of decompression sickness, Doppler "gas volume," and tissue ratio. The final set of relationships is shown graphically in figures 13 through 16. DCS can be described by the equation

$$\%DCS = a (Volume_{gas})^b \quad (11)$$

where $a = 11.95$

$$b = 0.60$$

and the correlation coefficient $r = 0.86$.

Direct correlation between the "gas volume" and the percent of subjects with gas bubbles was poor (figure 17).

D. Modified C-V Model

In the context of the model presented in the appendix, the tissue gas phase will develop with time as the system moves toward equilibrium, that is, dissolved inert gas and a gaseous phase divided according to Henry's law. Thus, there does not exist a single time-independent TR that is optimal for all decompression situations.

This is analogous to the original Haldane model with its multiple-tissue half times and allowable supersaturations. The system can be optimized to reduce the incidence of decompression sickness, but always admits to the possibility of its occurrence. Fortunately, the hypobaric situation involves a decompression only to one pressure in contrast with saturation decompression in deep-sea diving; in the latter case, the pressure is constantly reduced keeping the system under a constant supersaturation condition. Since decompression from deep saturation can be accomplished, equal or better success can be expected in the hypobaric environment.

E. Isobaric Counter Transport Mechanisms

If one closely observes the patterns of the appearance of Doppler-detectable gas bubbles, one notes the usual delay from the instigation of decompression. Gas phase formation (and Doppler-bubble release) usually occur early following the reduction in ambient pressure as can be seen in figure 2 or 3. This can be contrasted with the formation-and-release curves of figures 18 and 19 for hypobaric situations. These seem to be inordinately long especially when one considers that the subjects are breathing against a medium **totally devoid** of nitrogen.

In 1971, Blenkarn found that breathing one gas mixture and suddenly switching to another could under some conditions produce tissue gas phase formation in the absence of decompression (Blenkarn, et al., 1971). A similar situation of lesions arose when subjects were breathing one mixture while seated in another (Lambertsen and Idacula, 1975). In both cases, the effect was shown to be the result of certain combinations of solubilities and diffusivities such that the sum of the tissue partial pressures exceeded the ambient pressure. This would result either in spontaneous nucleation or the growth of those already existing in tissue (preformed nuclei). The effect is termed "counter transport" by D'Aoust, and it has been extensively reviewed by D'Aoust and Lambertsen (1982).

In the mathematical treatment of the transport of gas into and out of gas pockets (bubbles), Van Liew and Hlastala (1969) started with the Fick equation

$$dR/dt = (\beta_i \alpha_i P_{amb}) (1 - P_{ai}/P_{gi}) (1 + R/\theta_i) \quad (12)$$

where

$$\theta_i = [\beta_{bi} Q] / (\beta_{ti} \alpha_i) \quad (13)$$

where V is the bubble volume at STP, β_i and α_i are the coefficients of solubility and the diffusion coefficient of the i th gas, respectively; A is the surface area of the (spherical) bubble, P_i is the pressure of the i th gas dissolved in the tissue at a radial distance of r where Q is the effective tissue perfusion (it contains a multiplier factor for end-capillary perfusion); P_{ai} and P_{gi} are the pressures of the i th gas in the arterial blood and tissue, respectively; and β_{bi} and β_{ti} are the solubilities of the i th species in blood and tissue, respectively. The three terms in equation 12 are, respectively, from left to right:

- constants specific to the i th gas species
- partial pressure of the i th gas in the bubble and blood and tissue
- the reciprocal of the bubble radius and constants specific to the i th gas species

Hills (1977b) presented a similar situation in which different gas species with solubilities and diffusivities as above were separated by a tissue diffusion barrier of area A and thickness x . In this model, the gas phase could form in the blood vessels (if the sum of the partial pressures exceeded ambient). The model would allow the slow growth of a tissue gas phase (either formed by decompression or growing from micronuclei) tissue gas phase.

In this model, the flux ϕ for gas 1 is

$$\phi_1 = A\alpha_1\beta_{t1}(P-p_1)/x = Q\beta_{b1}P_1 \quad (14)$$

and for gas 2

$$\phi_2 = A\alpha_2\beta_{t2}p_2/x = Q\beta_{b2}(P-p_2) \quad (15)$$

Substituting

$$\phi = A/xQ,$$

$$n_1 = \alpha_1(\beta_{t1}/\beta_{b1}), \text{ and}$$

$$n_2 = \alpha_2(\beta_{t2}/\beta_{b2})$$

Thus,

$$p_1 + p_2 - P = \Phi P(n_1 - n_2)/(1 + \Phi n_1)(1 + \Phi n_2) \quad (16)$$

Hills noted that there would be supersaturation of venous blood, and the gas phase would grow, if

$$n_1 \geq n_2 \quad (17)$$

In both presentations of counter transport, steady-state growth of "silent," preformed gas phase will occur if the characteristics of the **incoming** gas species with respect to the exiting gas species are

$$\beta_1 W_1^{-1/2} \geq \beta_e W_e^{-1/2} \quad (18)$$

In the hypobaric situation, tissue nitrogen is gradually being eliminated (either by perfusion or by gas bubble formation and dispersion by the venous blood) so the tissue gas phase is not stable for long periods. In table VI are listed the solubilities and diffusivities of various gas used in hyper- and hypobaric environments (cf. Van Liew, 1968).

Table VI
Diffusivities and Solubilities

<u>Gas</u>	<u>Solubility \times Diffusivity ($\times 10^8$)</u>
O ₂	1.7
CO	1.3
A	1.87
N ₂	1.0
H ₂	1.94
He	1.44

It is evident from this compilation that the solubility-diffusivity ratio for oxygen to nitrogen is 1.7. This would indicate that, in some situations, it might be possible for oxygen to enter dysbarogenic tissue gas bubbles at a rate faster than nitrogen would exit. Oxygen would be continuously removed via cell metabolism, and any bubbles generated would naturally be small.

In figure 20, we see the Doppler-detectable bubbles (counts/min) determined by D'Aoust and Lambertsen in sheep when isobaric gas switches (three cases in which nitrogen was switched to helium and one case with nitrogen to neon) were performed. In these examples, the rapid rise in number of bubbles shortly after decompression is absent; the pattern is one of a rather long onset (1 to 3 hours) and then a rise and fall of bubble count. This is more congruent with the hypobaric case in which the gas phase generated by decompression can be augmented by the influx of oxygen; figure 19 illustrates several cases from NASA data of Doppler gas volume in a decompression with a ratio of 1.68. The rapid initial rise seen in decompressions of nitrogen to nitrogen (air to air) is seen in figures 3 and 4. The effect cannot be attributed solely to the fact that "slow tissues" are involved since a similar pattern is seen when nitrogen saturation decompression is effected without breathing against pure oxygen (figure 21, from Eckenhoff et al., 1986). In this latter case, the rise in bubble number is immediately after decompression, and the Spencer Doppler grades are low (Grade III maximum on any subject) in contrast to the numerous and persistent cases of Grade IV often seen in hypobaric situations.

VI. IMPLICATIONS

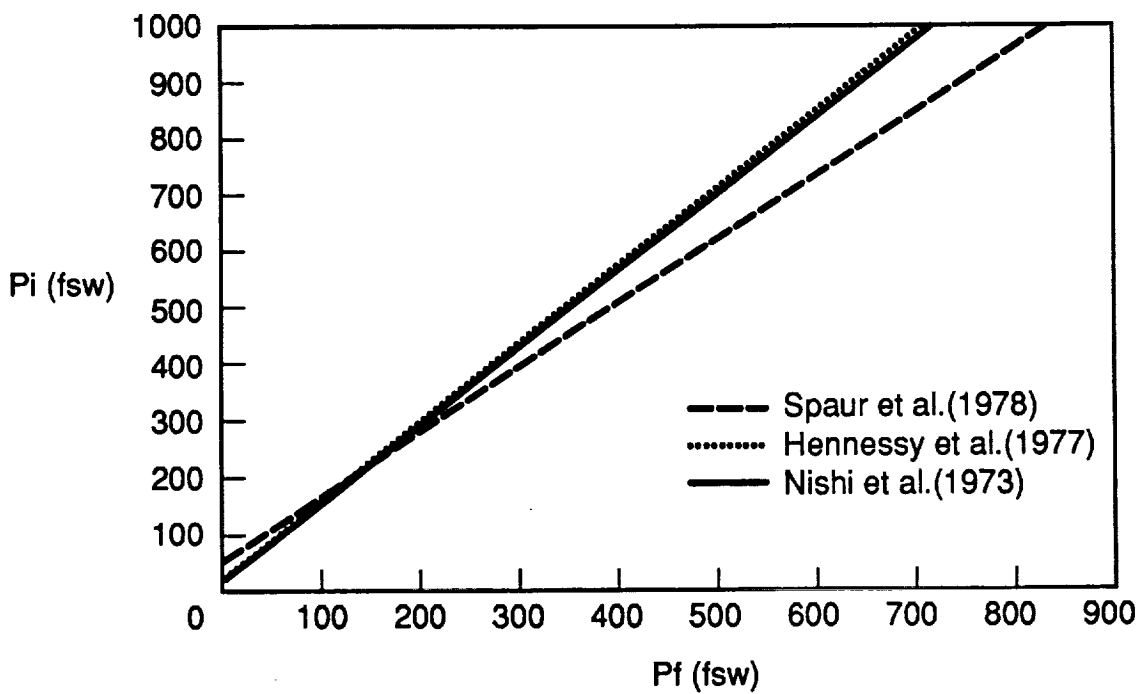
Those processes that **modify the number of tissue micronuclei** (e.g., negative pressure, tribonucleation [viscous adhesion], etc.) will have the greatest influence as specific individual variables on the decompression outcome.

Since the **lower extremities** appear to be the **most susceptible** to forces that **generate tissue gas micronuclei**, they are among the greatest generators of gas volume. They are the regions of the human body most susceptible to altitude decompression sickness (Waligora et al., 1984).

It is tempting to postulate that there will be a **reduction of tissue micronuclei** in microgravity as compared to unit gravity. A reduction in the forces (e.g., negative pressure, shock waves, tribonucleation) normally experienced during walking in unit gravity will result in zero-g in the reduction of tissue gas micronuclei in the lower extremities. This could contribute to a reduced reported incidence of decompression sickness among the astronauts during extravehicular activity (EVA); the reported EVA incidence is considerably less than would be expected from laboratory

studies performed under unit gravity. (This could be investigated with decompression of subjects subjected to several days of bed rest.)

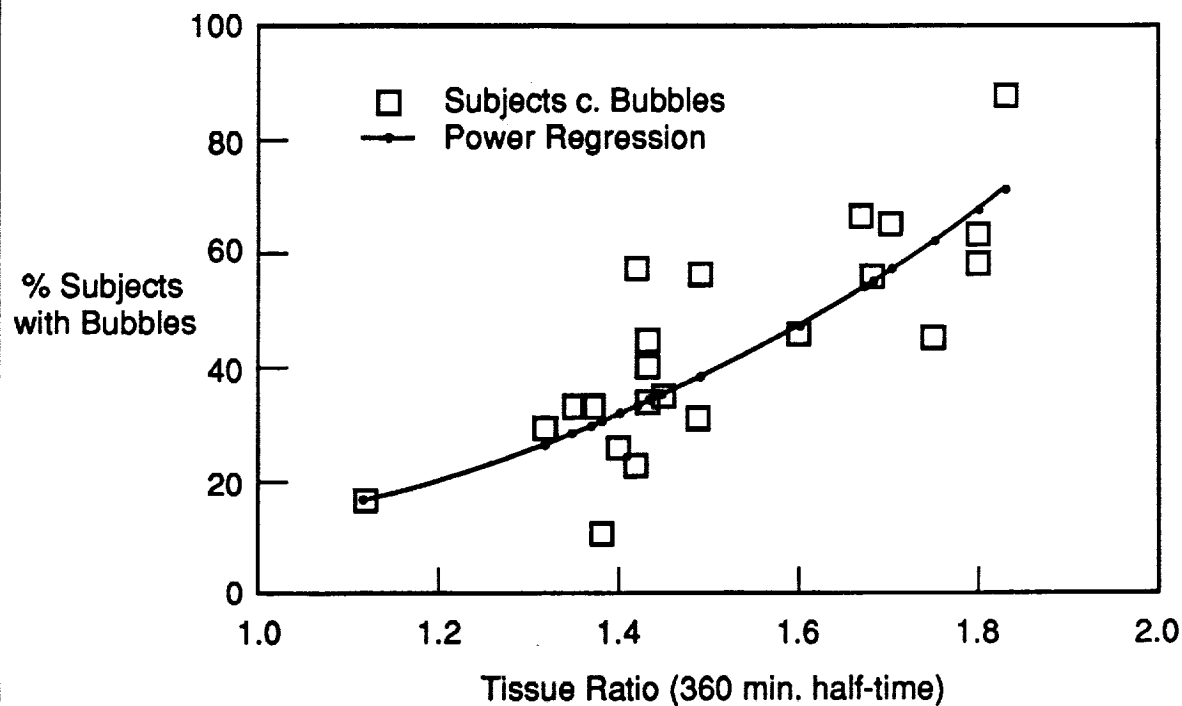
Decompression from Saturation Conditions Air and Heliox Diving



X101211

Figure 1

Percent Subjects with Doppler-detectable Gas Bubbles vs. Tissue Ratio

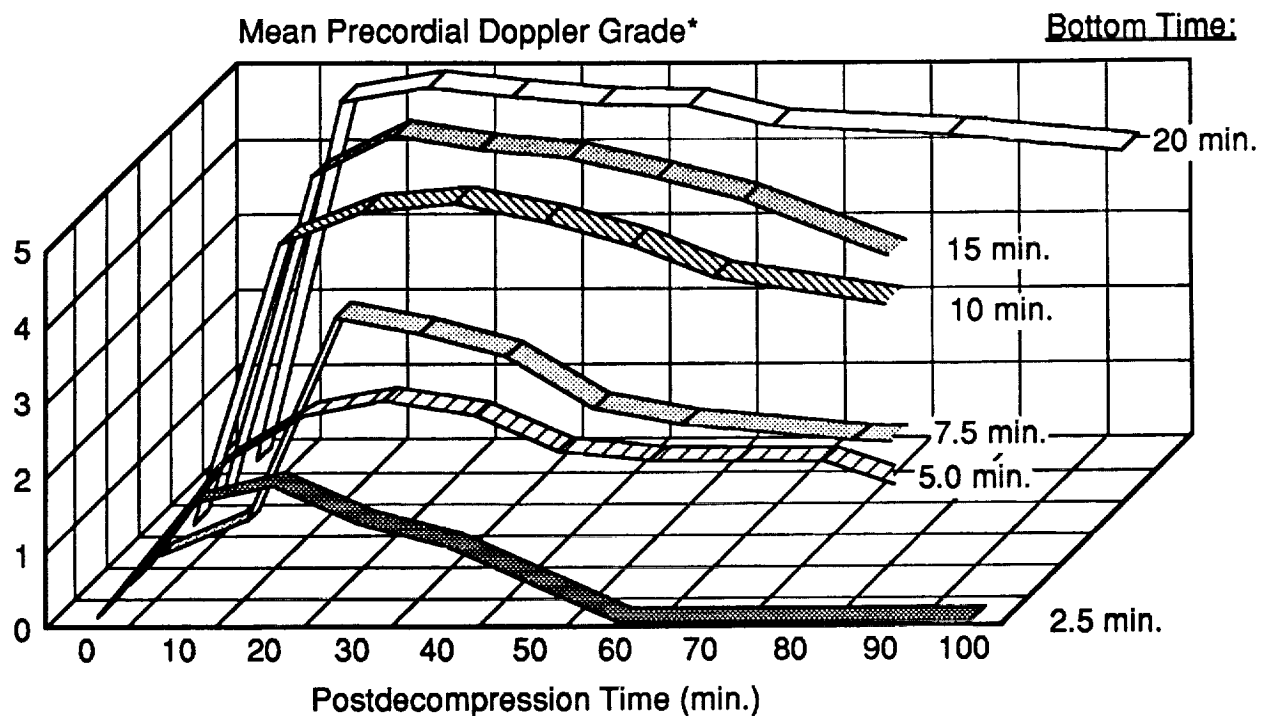


From the data of Conkin et al. (1987)

X101212M

Figure 2

Growth and Decay of Precordially Monitored Doppler Bubbles



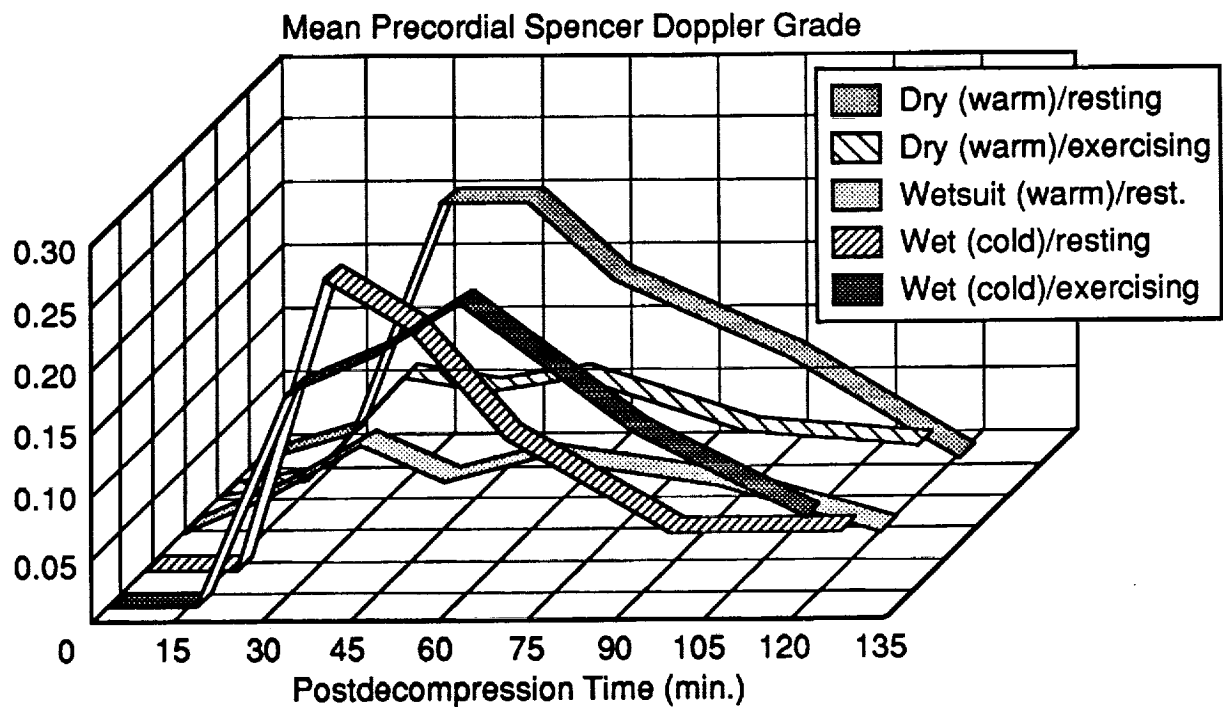
* Modified system of Smith and Powell;
cf. Powell and Spencer (1980)

Subjects: Sheep

X101213M

Figure 3A

Decompression Stress Under Varying Environmental Conditions

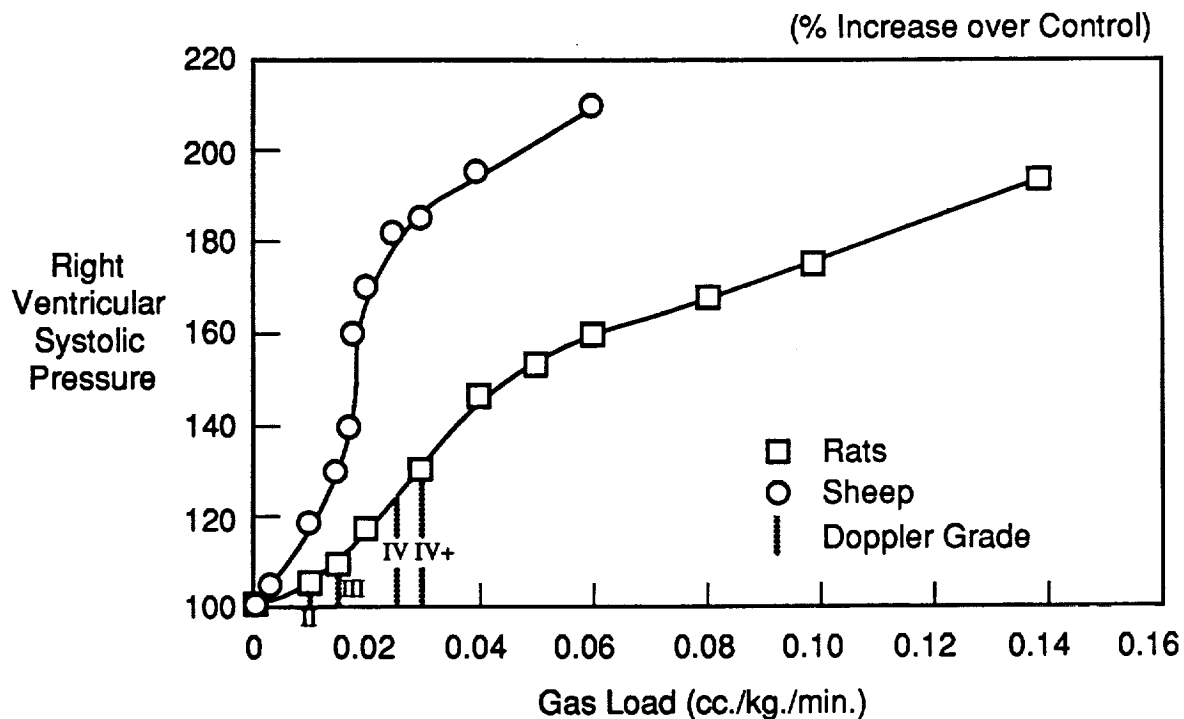


Dive: 100 FSW/50 min.
Water temperature: 18° C

X101214M

Figure 3B

Right Ventricular Systolic Pressure vs. Injected Gas Loads

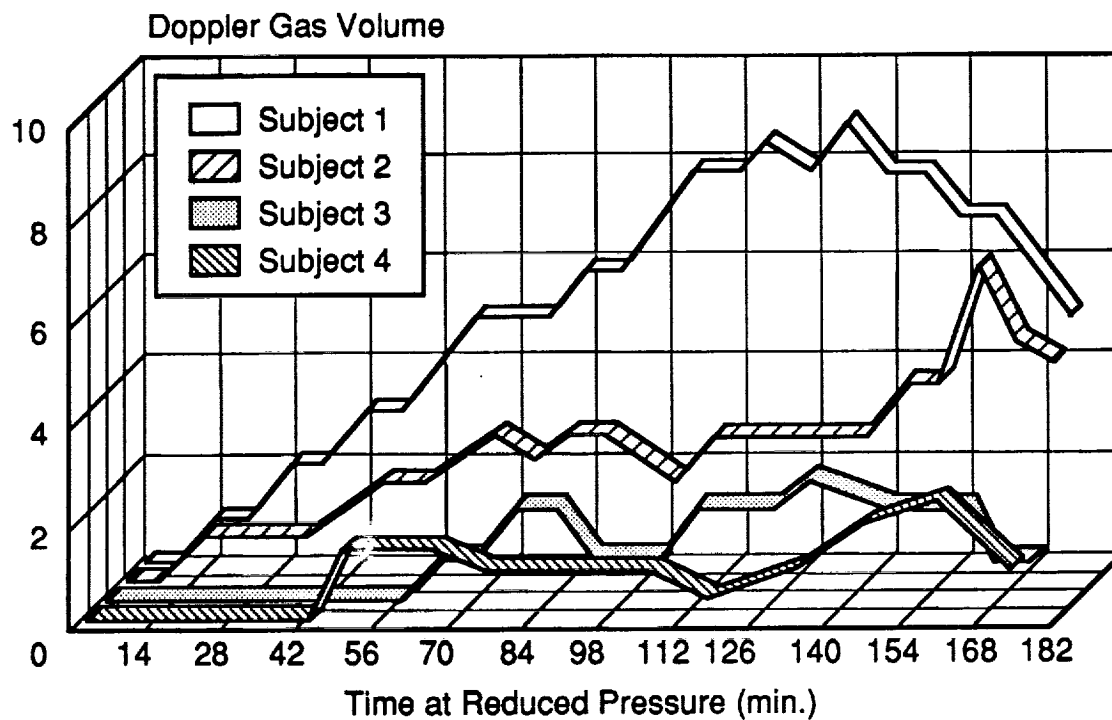


From Powell and Spencer (1980)

X101215M

Figure 4

Time-Intensity Decompression Stress Doppler Gas Volume vs. Time

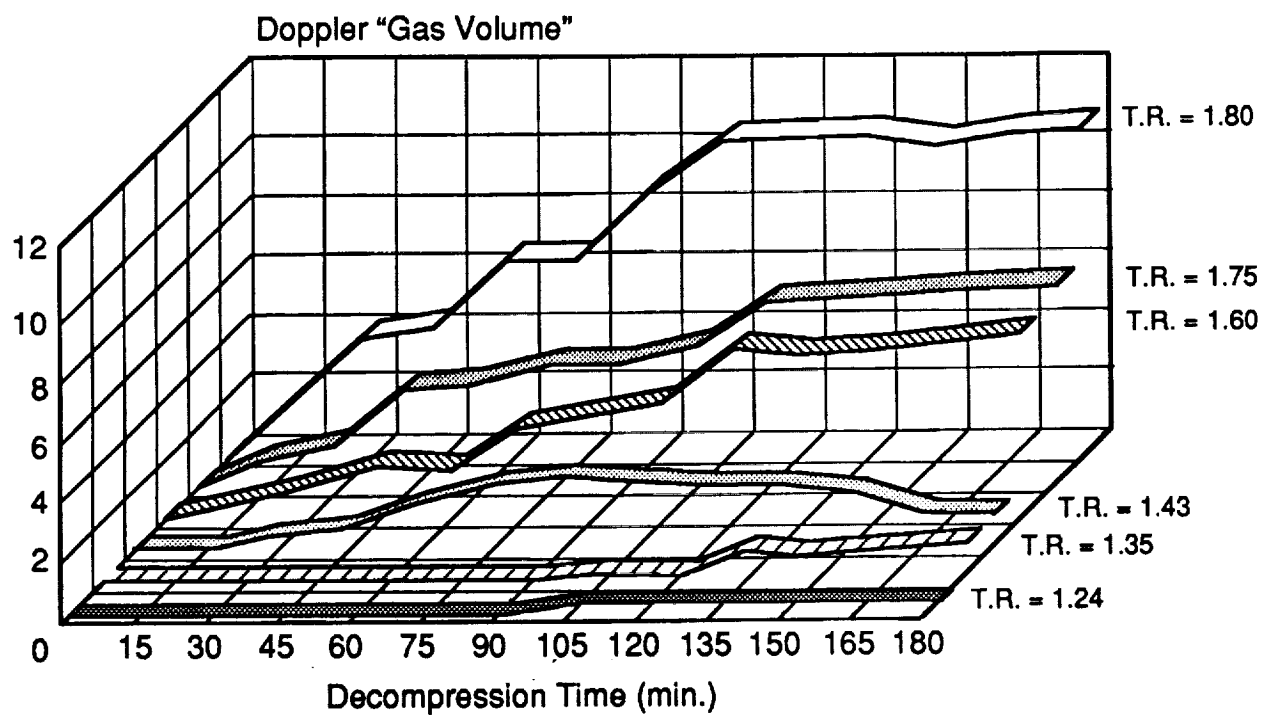


* For conversion of Spencer Doppler grade to gas volume, see text.

X101216M

Figure 5

Generated "Gas Volumes" at Varying Tissue Ratios

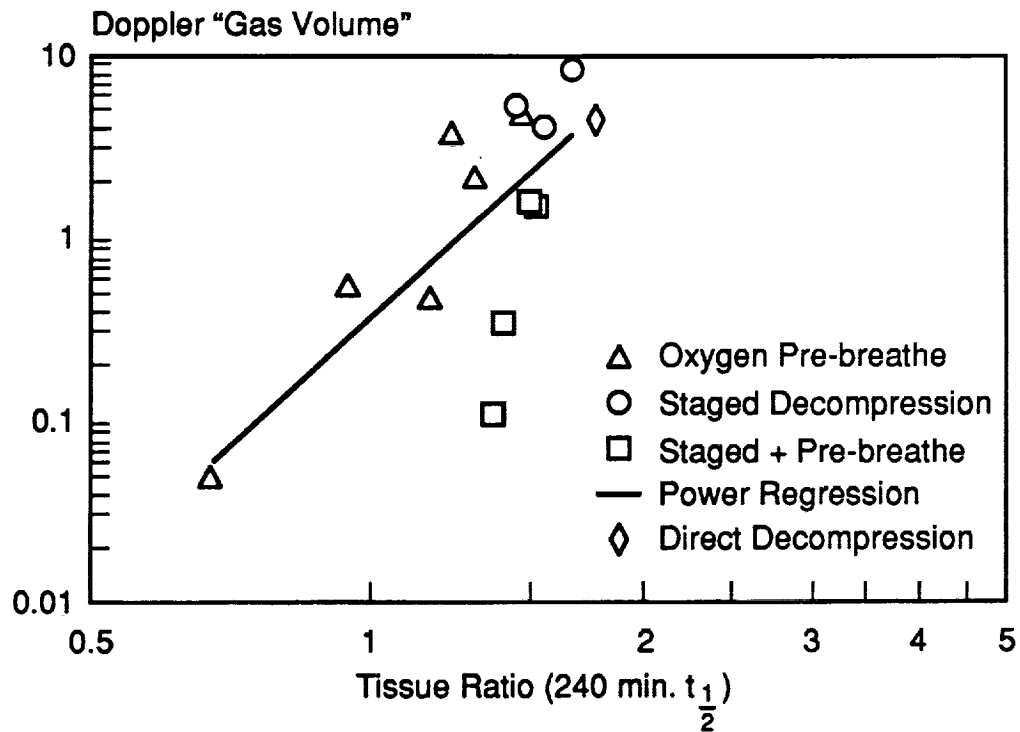


From the data of Conkin et al. (1987)

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Figure 6

Doppler "Gas Volume" vs. Tissue Ratio: Staged and Pre-breathe Decompressions

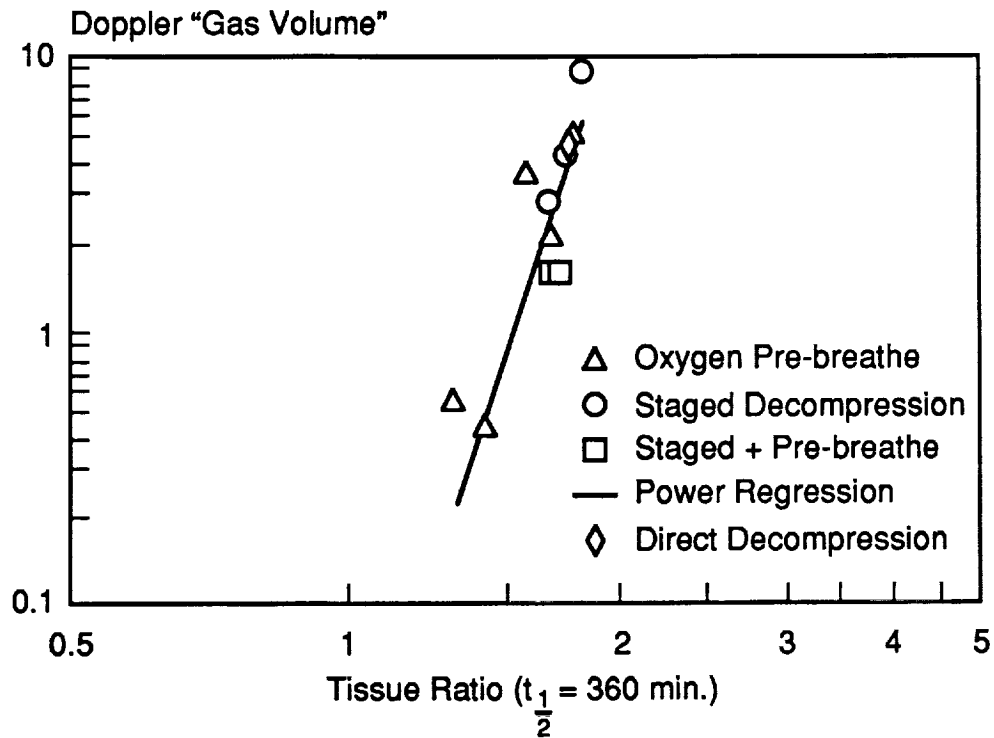


From the data of Conkin et al. (1987)

X101218M

Figure 7

Doppler "Gas Volume" vs. Tissue Ratio: Staged and Pre-breathe Decompressions

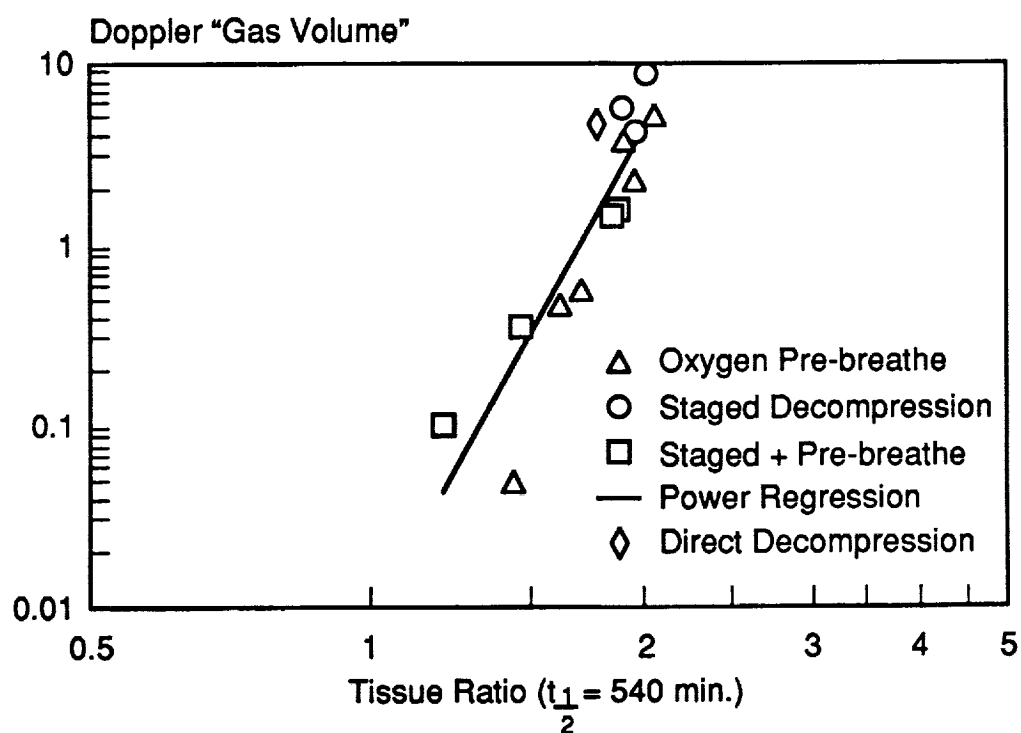


From the data of Conkin et al. (1987)

X101219M

Figure 8

Doppler "Gas Volume" vs. Tissue Ratio: Staged and Pre-breathe Decompressions

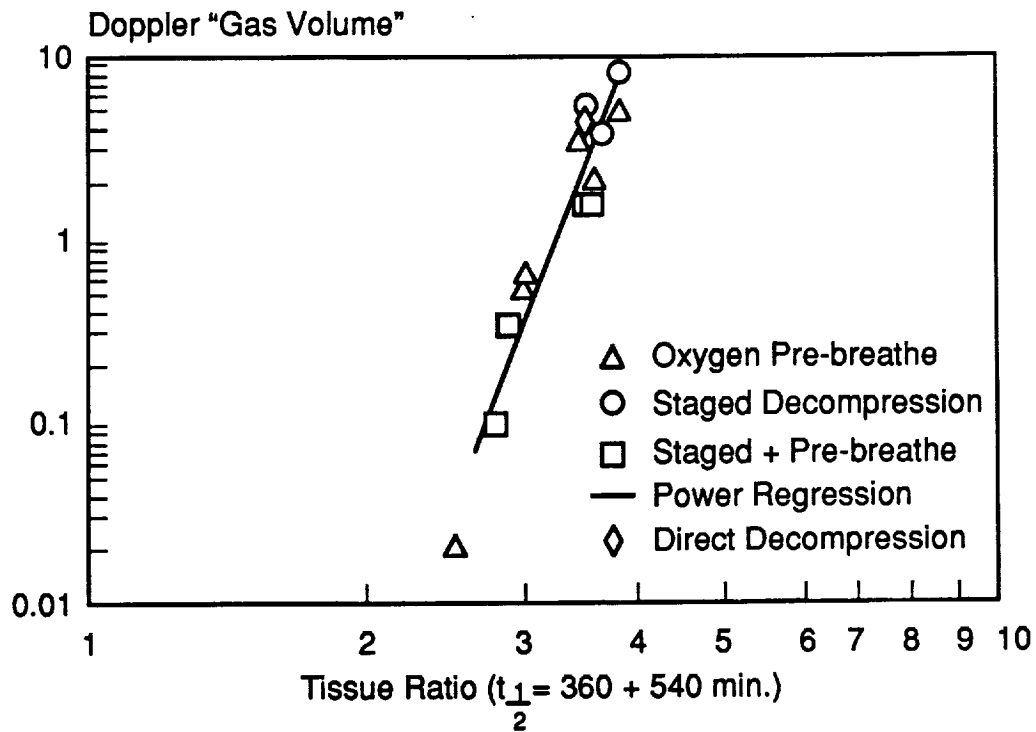


From the data of Conkin et al. (1987)

X101220M

Figure 9

Doppler "Gas Volume" vs. Tissue Ratio: Staged and Pre-breathe Decompressions

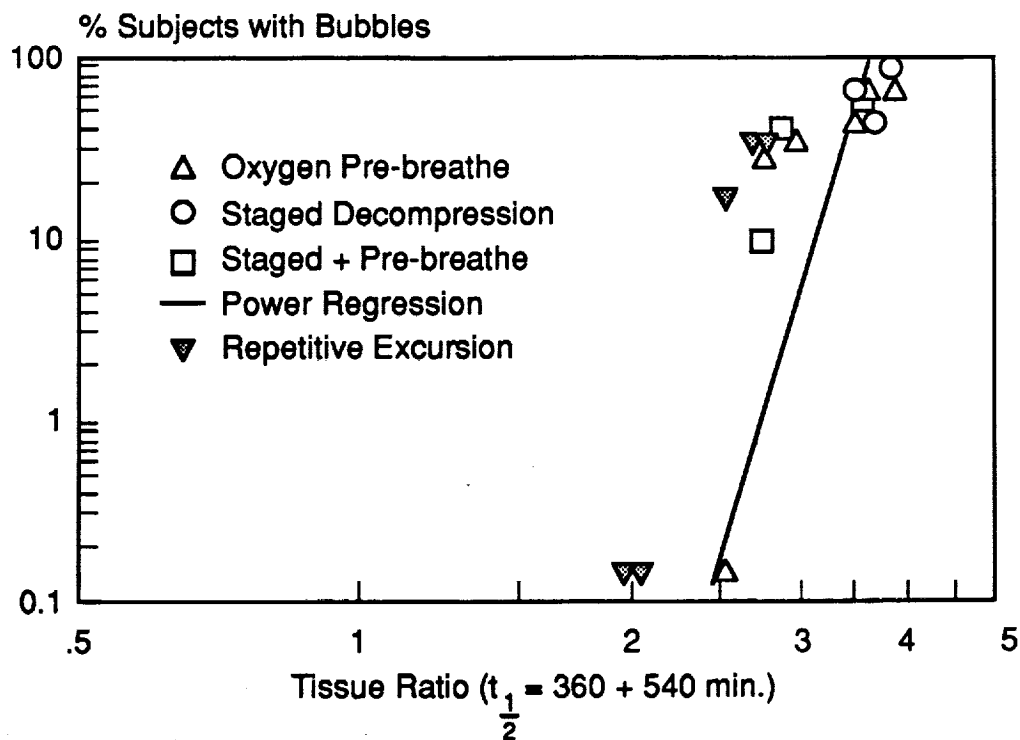


From the data of Conkin et al. (1987)

X101221M

Figure 10

Percent Subjects with Doppler Gas Bubbles: Staged and Pre-breathe Decompressions

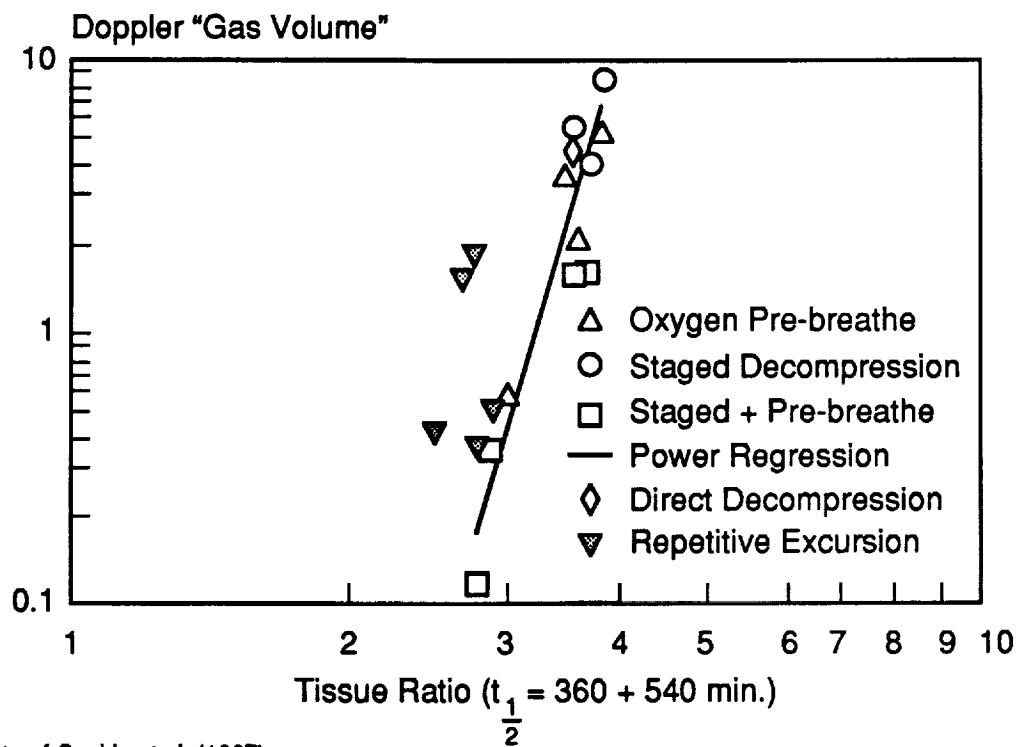


From the data of Conkin et al. (1987)

X101222M

Figure 11

Doppler "Gas Volume" vs. Tissue Ratio: Staged and Pre-breathe Decompressions

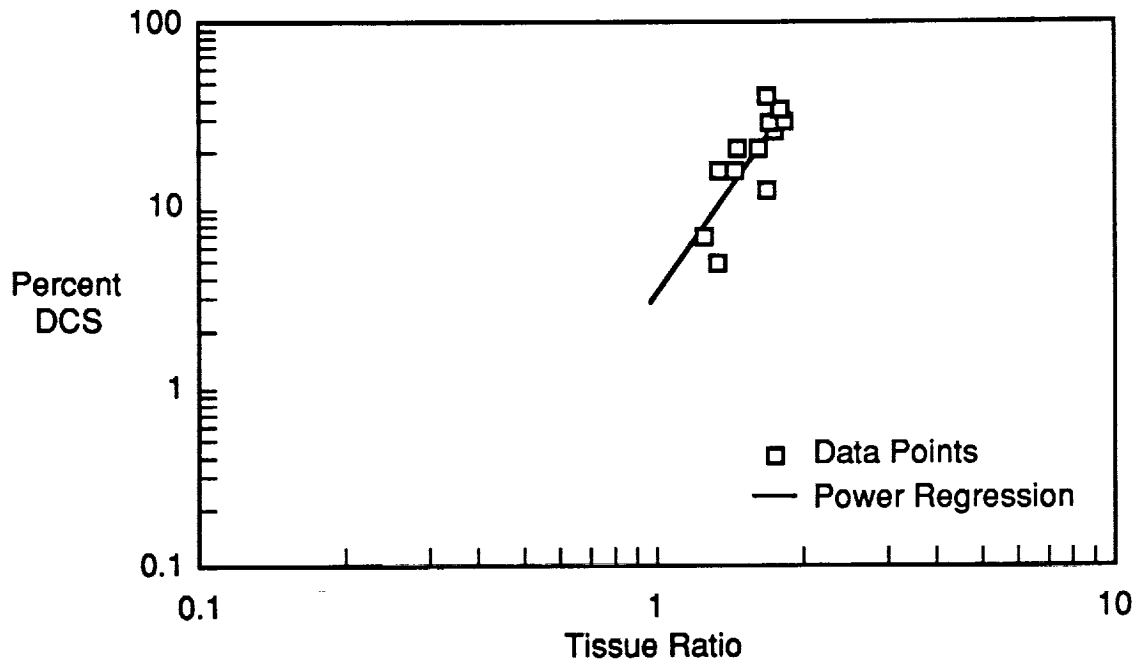


From the data of Conkin et al. (1987)

X101223M

Figure 12

Percent DCS vs. Tissue Ratio (180-minute test)

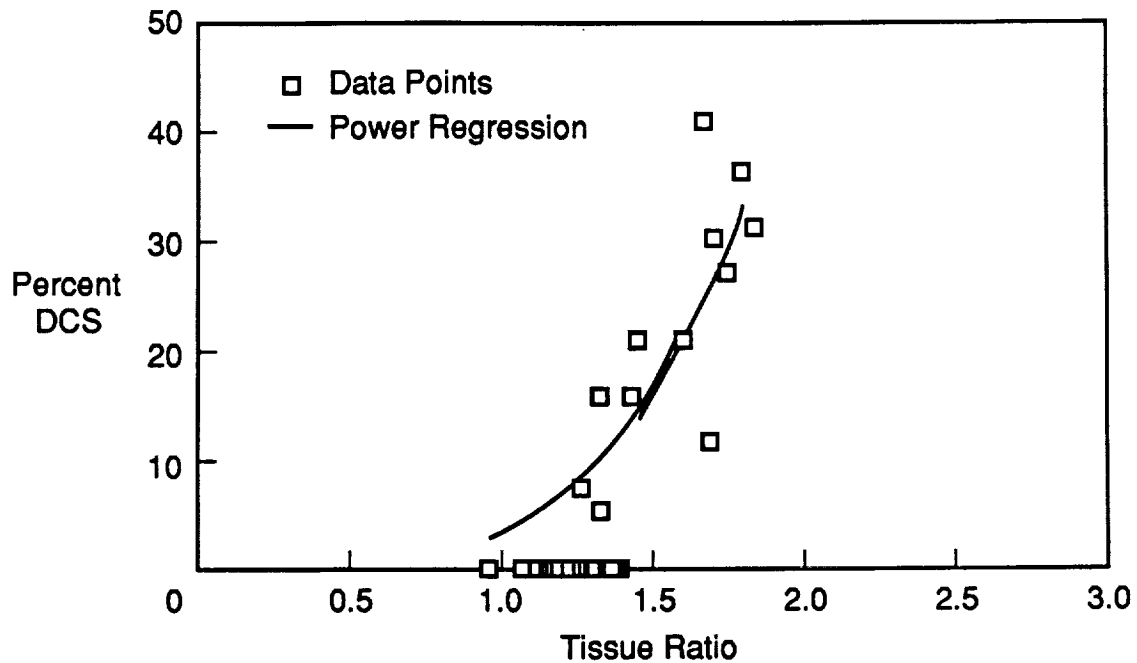


(USAF/SAM and NASA data)

X101224M

Figure 13

Percent DCS vs. Tissue Ratio (180-minute decompression)

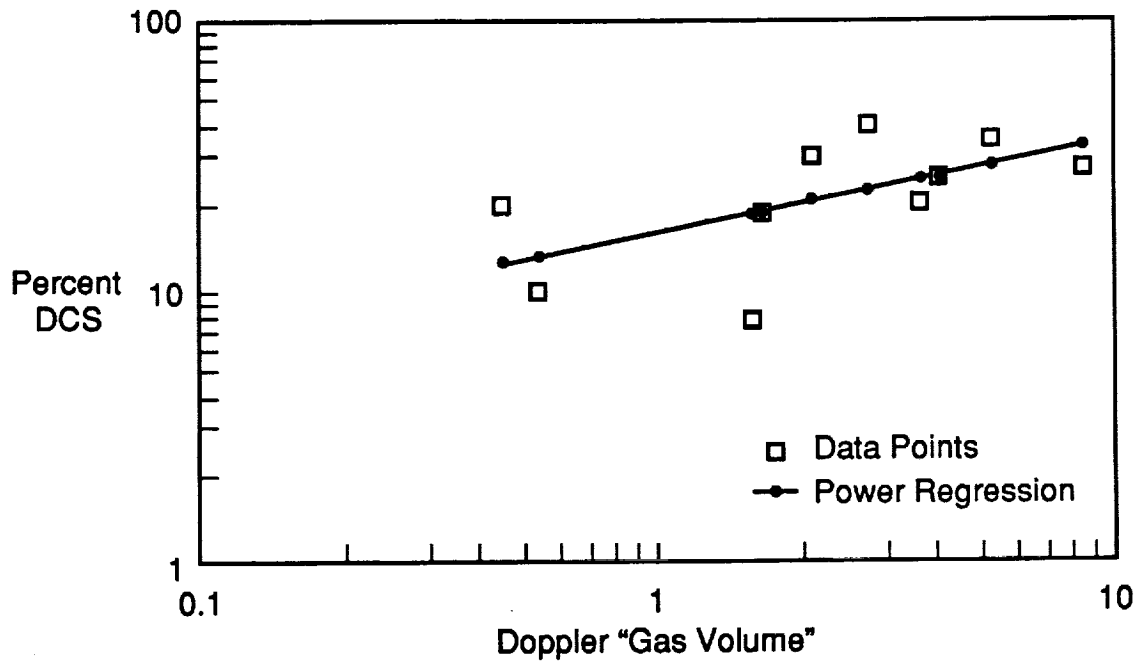


from the data of Conkin et al. (1987)
(USAF/SAM and NASA data)

X101225M

Figure 14

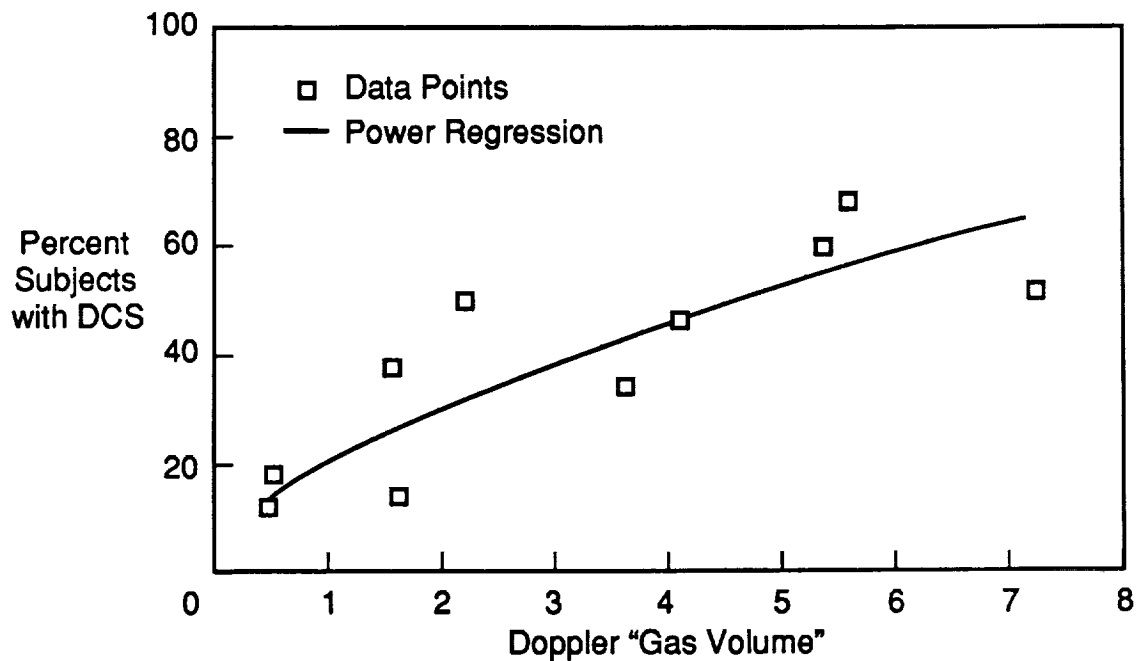
Percent DCS vs. "Gas Volume" (180-minute test)



X101226M

Figure 15

Percent DCS vs. Doppler "Gas Volume" (180-minute decompression)

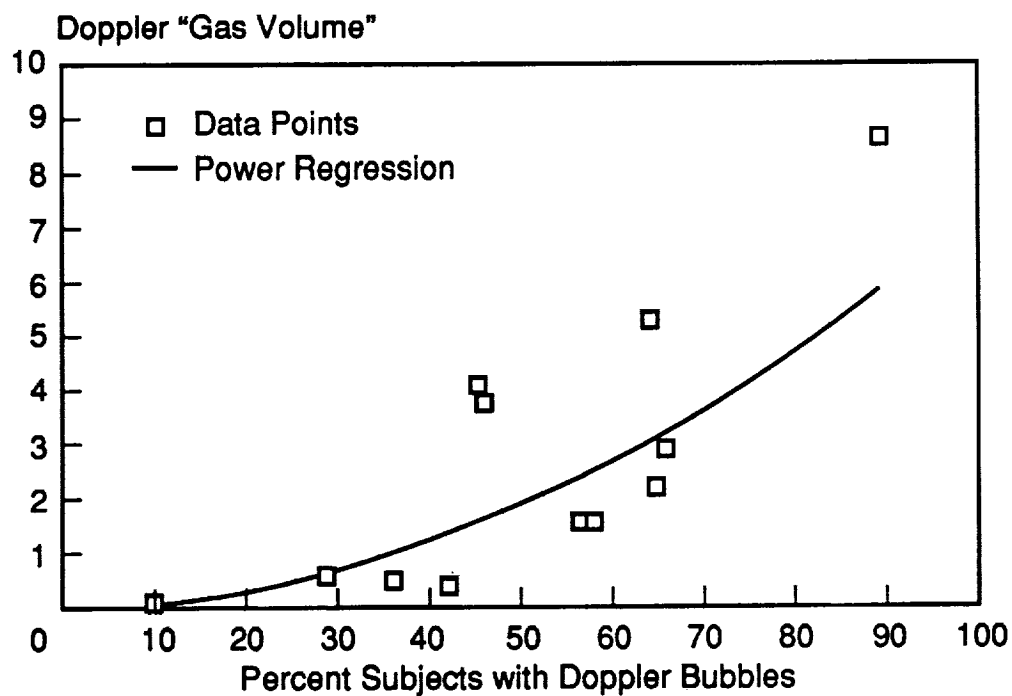


from the data of Conkin et al. (1987)

X101227M

Figure 16

Doppler "Gas Volume" vs. Percent Subjects with Doppler-detectable Gas Bubbles

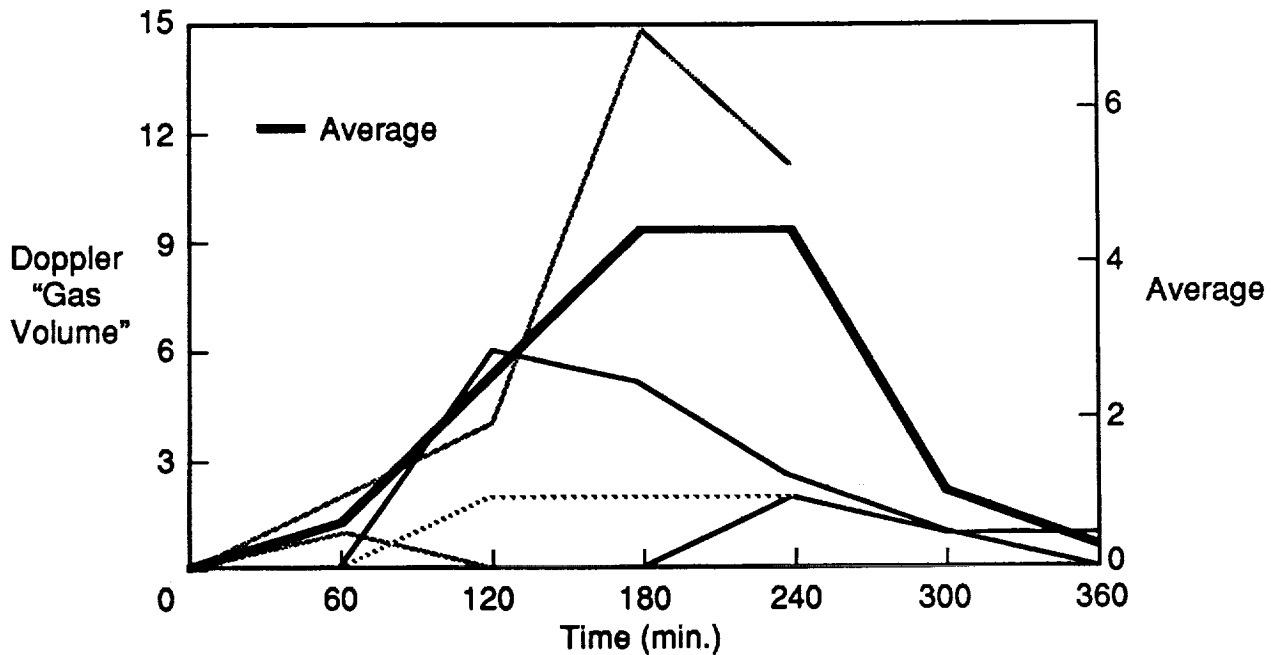


from the data of Conkin et al. (1987)

X101228M

Figure 17

Doppler-detectable Gas Bubbles During Hypobaric Decompression



Tissue Ratios = 1.12 and 1.24
Nitrogen to Oxygen

X101229M

Figure 18

Doppler-detectable Gas Bubbles During Hypobaric Decompression

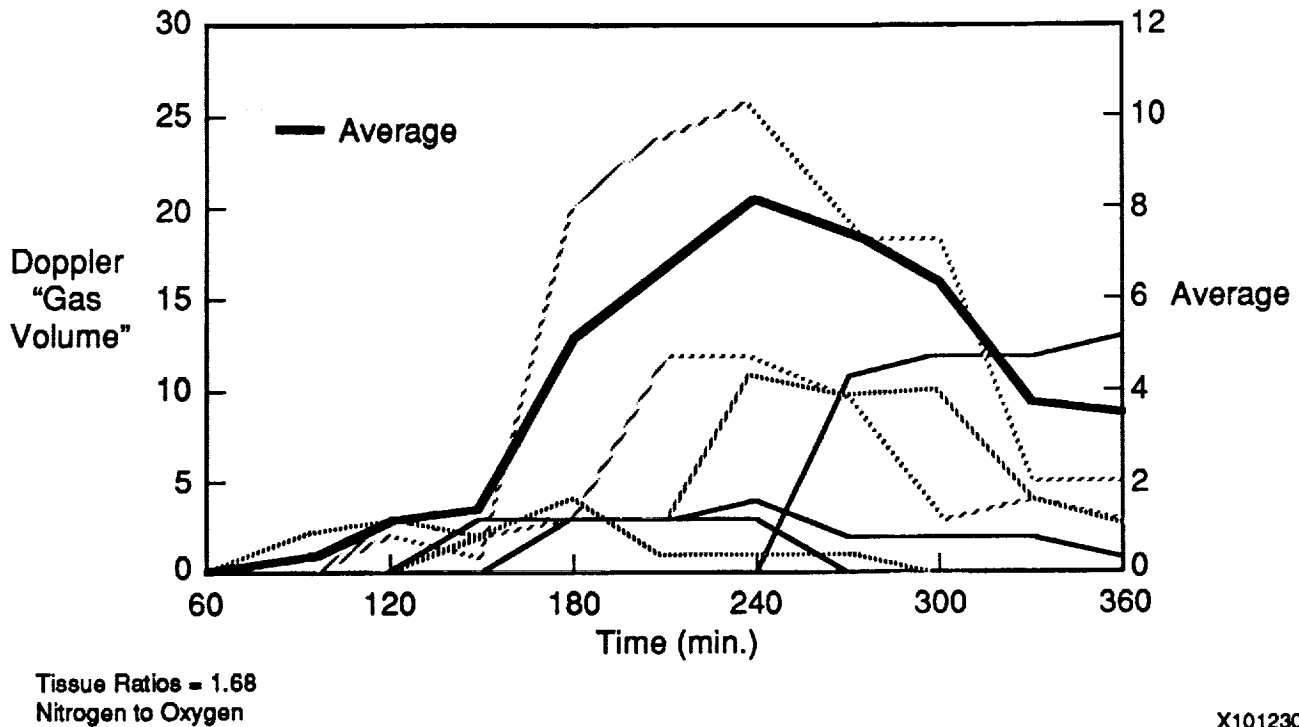
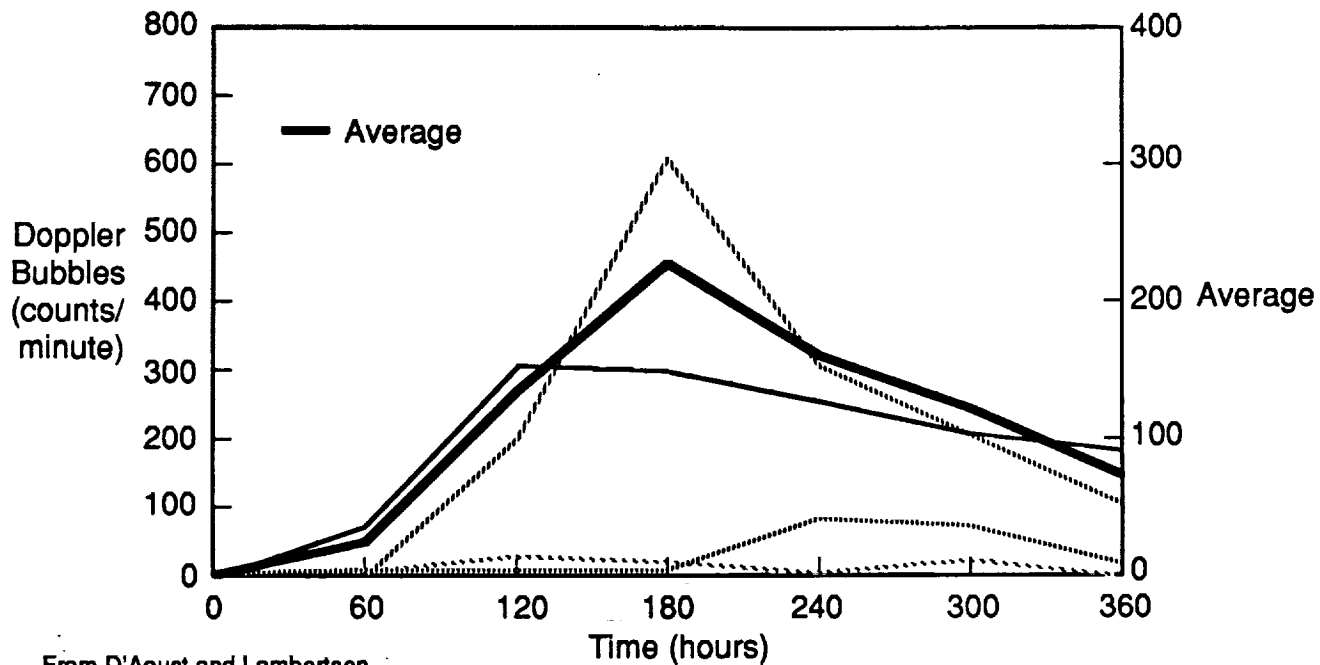


Figure 19

Isobaric Counter Transport and Doppler-detectable Gas Bubbles

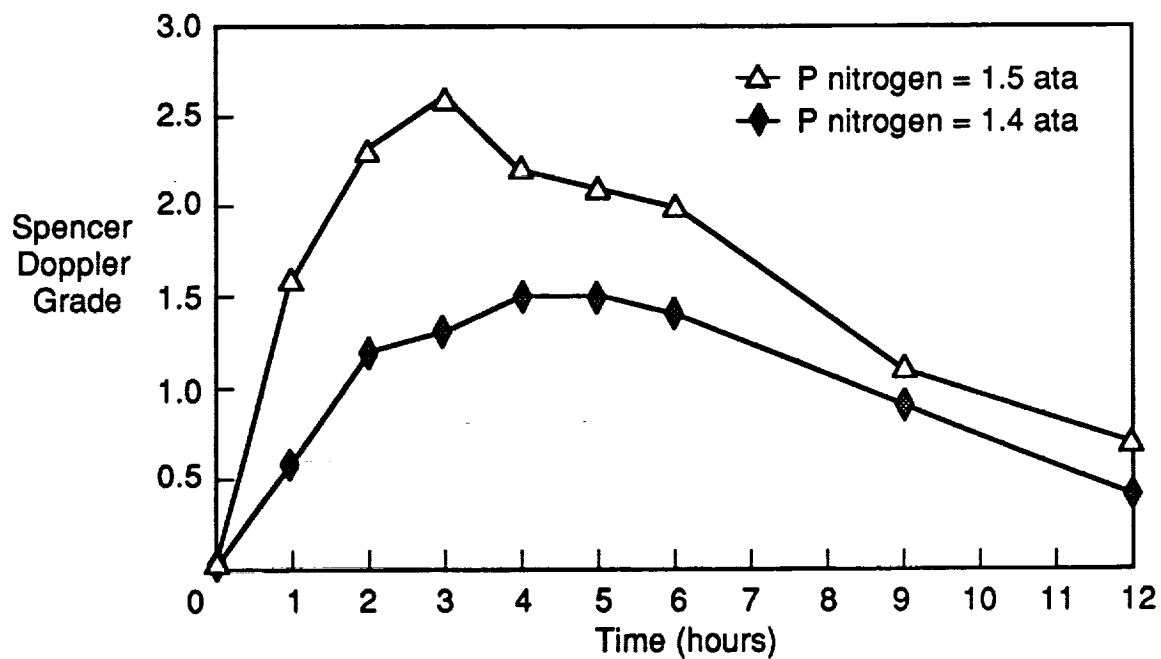


From D'Aoust and Lambertsen
in: Physiology & Medicine of Diving
(Eds. Bennett & Elliott) 1982

X101231M

Figure 20

Direct Ascent from Air Saturation



From Eckenhoff, et al.
Undersea Biomed. Res., 1986

X101232M

Figure 21

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APPENDIX A

Modified Critical Volume Hypothesis

A. Decompression of a Saturated Liquid

Let us consider the case where a volume of a liquid that is saturated with an inert gas (solubility = β) is enclosed in a container with a gas-tight piston as a lid. The liquid is saturated at a pressure P_i and an external pressure is applied to the piston to retain the inert gas in solution. If the external pressure were suddenly reduced to pressure P_f , a gas phase would form in the vessel, and the piston would eventually rise to produce a volume V in accordance with Henry's law.

Hills (1966) originally showed that a mass balance for the process could be given by the following equation which accounts for equal moles of gas on both sides of the equation.

$$\beta P_i = \beta P_f + VP_f \quad (1A)$$

Dividing by β gives

$$P_i = P_f + (V/\beta) P_f \quad (2A)$$

rearranging terms gives

$$(P_i/P_f) = (1/\beta) + 1 \quad (3A)$$

which would allow one to compute the solubility β of the gas by measuring the **total released volume** at each P_i/P_f .

A plot ($y = mx + b$) would give the slope $m = (1/\beta)$ and the intercept $b = 1$ if **no inert gas-saturated liquid was lost** from the apparatus and if **supersaturation did not occur**. This is graphically depicted in figure A-1.

B. Gas Volume With Supersaturation of the Liquid

If the volume of liquid should be supersaturated following the decompression, a lesser volume V of gas would be released. This would be given by the following where σ is the degree of supersaturation.

$$P_i \beta (1 - \sigma) = (1 - \sigma) P_f \beta + VP_f \quad (4A)$$

Rearranging terms as before in equation (3A) gives

$$P_i/P_f = [1/(1 - \sigma)][V/\beta] + 1 \quad (5A)$$

If one were to determine the solubility from the measurement of the released volume V , the computed value would be too small by $1/(1 - \sigma)$.

If one waits a sufficiently long time, the supersaturation will be reduced; indeed, at time = ∞ and $\sigma = 0$, equation (5A) will be reduced to equation (3A). This is shown graphically in figure A-2.

C. Decompressions With Loss of Saturated Fluid

If some fraction λ of the saturated fluid is withdrawn, the volume of gas that will be produced will be less. The mass balance equation for this is

$$P_i \beta (1 - \lambda) \beta P_f + V P_f \quad (6A)$$

The loss would be related to the percent volume λ of liquid removed.

With no supersaturation, but with loss λ

$$P_i / P_f = \frac{[V/\beta]}{1/(1 + \lambda)} + 1 \quad (7A)$$

The difference between equation (4A) and equation (7A) is that the volume of gas that one would collect in the latter case would be less and the collection **would not be time-dependent**. That is, waiting for a period of time would *not* increase the collected gas volume as the system would already be at equilibrium.

The case for a given degree of supersaturation but with varying amounts of loss of gas-saturated fluid is shown in figure A-3.

D. *In Vivo* Applications

For the case of *in vivo* decompression (whether from depth or from altitude), the end point will usually be determined by the presence or absence of decompression sickness. For the case where pain-only decompression sickness (the "bends") occurs, Hills (1966) proposed that a critical volume of gas V_c was released into the tendon and ligamentous tissues. A rearrangement of equation (3A) produced the familiar linear relationship between initial and final pressures, P_i and P_f , respectively, to result in limb-pain decompression sickness

$$P_i = ([V_c / \beta] + 1) P_f \quad (8A)$$

To produce a non-zero intercept, Hills introduced factors which included surface tension, tissue deformation pressure (Nims) and a pressure for critical nerve deformation (Inman and Saunders, 1944). Since any gas that would be present in the body would be subjected to small pressures in addition to the ambient, it is necessary to account for them in some manner. Hills did this by replacing P_f in equation (1A) with p_t , the pressure at the tissue level. In the manner in which the equations are here derived, this would be equivalent to the pressure exerted by the weight of the piston δ itself.

$$p_t = P_f + \delta$$

For the *in vivo* case, equation (1A) then becomes

$$S P_i = S (P_f + \delta) + V (P_f + \delta) \quad (9A)$$

The equation was not meant to explain events other than saturation decompression with a single step. We know from experiment that most of the inert gas remains in solution following decompression

(Powell, 1972b, 1973; Powell and Weydig, 1974; Powell, 1977; Powell, Spencer and von Ramm, 1982; Hills, 1978). Thus, combining equation (5A) with equation (7A) into (9A) produces

$$P_i = \frac{(V_c/\beta) + 1}{(1 - \sigma)(1 - \lambda)} [P_f + \delta] \quad (10A)$$

where σ and λ will vary from zero to unity. Therefore, in a plot of P_i vs. P_f , a plot that is used to illustrate the change of maximum allowable tissue pressure (M -value) reduction with pressure, the slope will contain the factors

$$m = [1/(1 - \sigma)(1 - \lambda)] [V_c/\beta] + 1$$

and the y-intercept will be

$$b = \{1/(1 - \sigma)(1 - \lambda)\} (V_c/\beta + 1) \delta$$

This is depicted in figure A-4.

For tissues of varying half times, the variation of slope can be explained by the increased value of both σ and λ . This simple physico-chemical model accords with the classical explanation that "fast" tissues can sustain a higher degree of supersaturation and also possess a high perfusion. Bühlmann's data indicates that extrapolation of the ascent limiting lines for tissues of various half times yields intercepts of different values. This can be explained by the variation in the gas washout factor λ , the degree of inert gas solubility β , or the variation in the "critical volume" V_c .

The common notion that "fast" tissues are highly perfused would seem to indicate that the principal cause of increased tolerance of these tissues is the difficulty of reaching a sufficient gas volume V_c in these regions.

Experimental studies with "faster" tissues have shown that the tissue gas phase forms slowly (Powell, 1972b), and that dissolved gas will be continually removed by the blood. The treatment presented here indicates that the "long half-time tissue" involved in the development of altitude decompression sickness can be described as one that has a very small loss factor and does not sustain a high degree of oversaturation; this derived concept accords with the classical treatment.

In reality, it is not a physical requirement that σ and λ remain constant within the tissues during the decompression phase. The smaller the amount of inert gas in physical solution (smaller value of σ), the greater will be the volume of tissue gas V produced and the smaller will be the loss of inert gas from the tissue via the blood. In living systems, two factors play a role with respect to the development of a tissue gas phase (and subsequent decompression sickness).

- (1) The blood perfusion is not constant within a given decompression period, a considerable degree of temporal inhomogeneity exists. Any variation in the degree of supersaturation will produce a change in the total amount of dissolved gas which can be removed and in the perfusion constant.

- (2) The degree of supersaturation is time-dependent, that is, $\sigma = f(t)$; given a sufficient period of time, any supersaturation will disappear. The total amount of gas which can be released into any microvolume then becomes a function of:
- the amount of dissolved tissue inert gas taken up during the compression phase (this puts the limit on potential tissue gas volume)
 - the amount which actually forms since there is a loss of dissolved inert gas as a result of perfusion

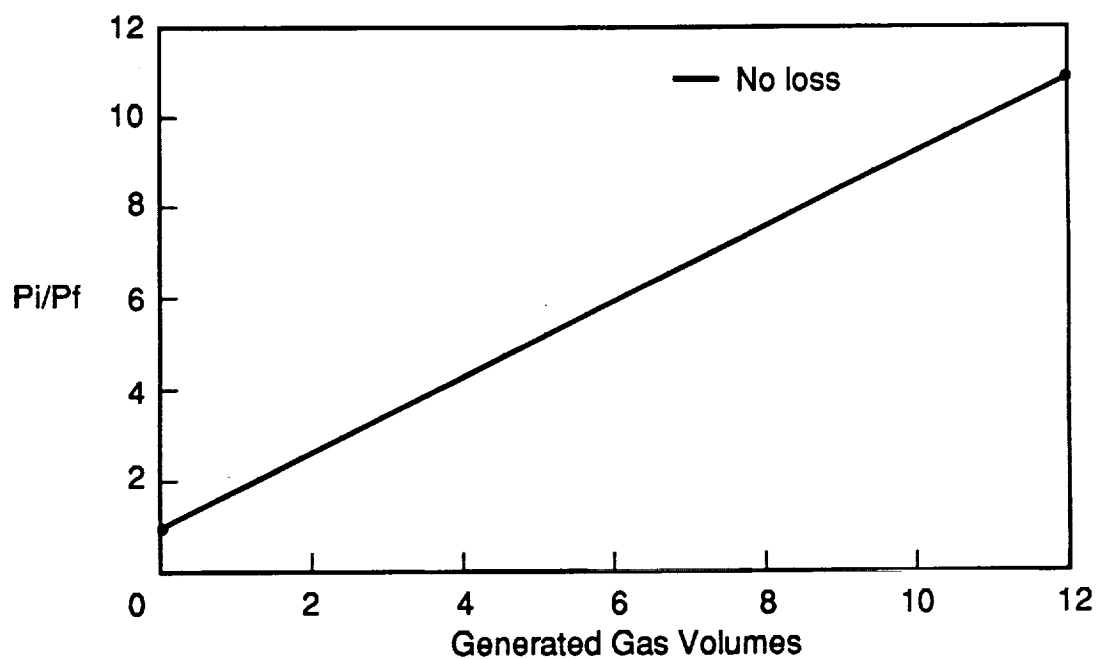
These are interrelated and the system can become decidedly nonrobust (Powell and Rogers, 1989). When systems have varying degrees of loss of dissolved inert gas and varying degrees of supersaturation, curves of the type in figure A-4 can be generated that strongly resemble the allowable ascent curves for tissue with differing half times (see Bühlmann, 1984, p. 28).

(3) The **primary variables** in the modified critical volume (or stoichiometric) model are the **degree of supersaturation** present during decompression in a given compartment (or microregion) of a tissue and the **perfusion**. From experiment, the degree of supersaturation is known to be generally quite high, probably $\sigma > 0.9$. It is postulated that the primary reason for the appearance of the signs and symptoms of decompression sickness is the variation in the number of tissue gas micronuclei, the agents responsible for the loss of supersaturation. These will modify both the volume of gas produced at the tissue level during decompression and the number of moles of gas lost from a microregion during decompression; the process is nonlinear. This concept is at variance with the ideas first proposed by Hills in which rapid phase transformation is assumed to occur.

Perfusion rates are generally constant over a long interval although they exhibit temporal perfusion heterogeneity. It has been suggested by Dunford (private communication) that **closure of a vessel is probably longer in poorly perfused tissues or regions**; this would lead to a higher probability, with time, of phase transformation in compartments with long half times. This is reflected in the increased difficulty of effecting a safe decompression when prolonged decompression times (that is, the need to invoke the long half-time compartments) are required.

Since dissolved inert gas is lost through perfusion during the decompression phase, the longer the interval between the pressure change and the initiation of gas phase formation, the greater the total volume V can become. The degree or intensity of the dcs problem(s) and the percentage of subjects developing problems should be inversely related to the time of onset. This has been found to be true for altitude dcs (Ferris and Engel, 1952).

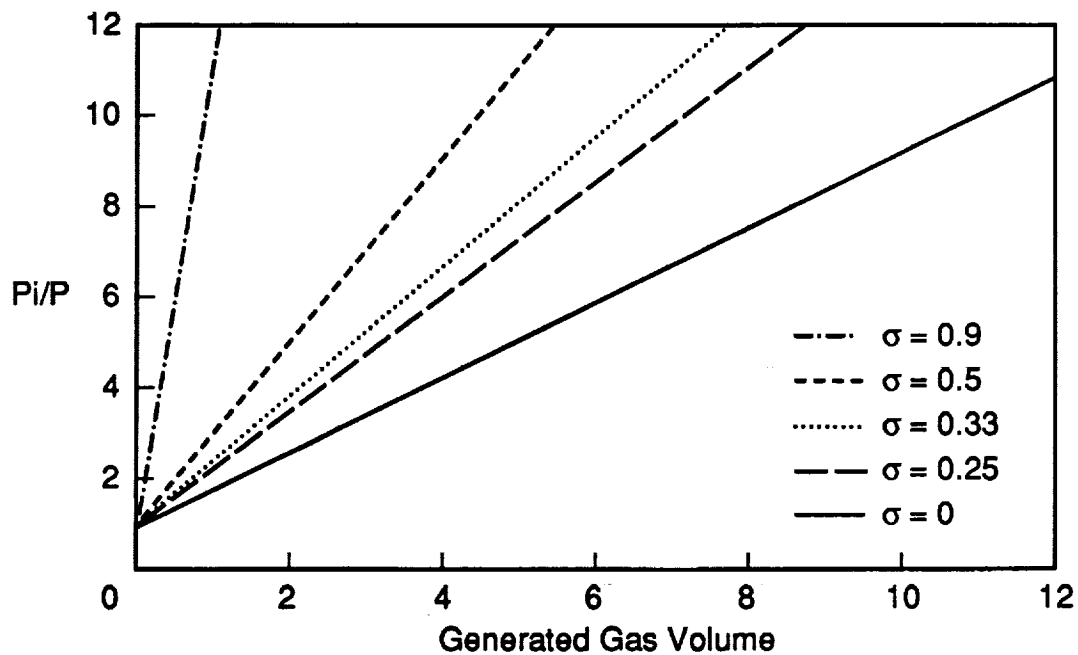
Gas Volumes Created with Decompression



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Figure A-1

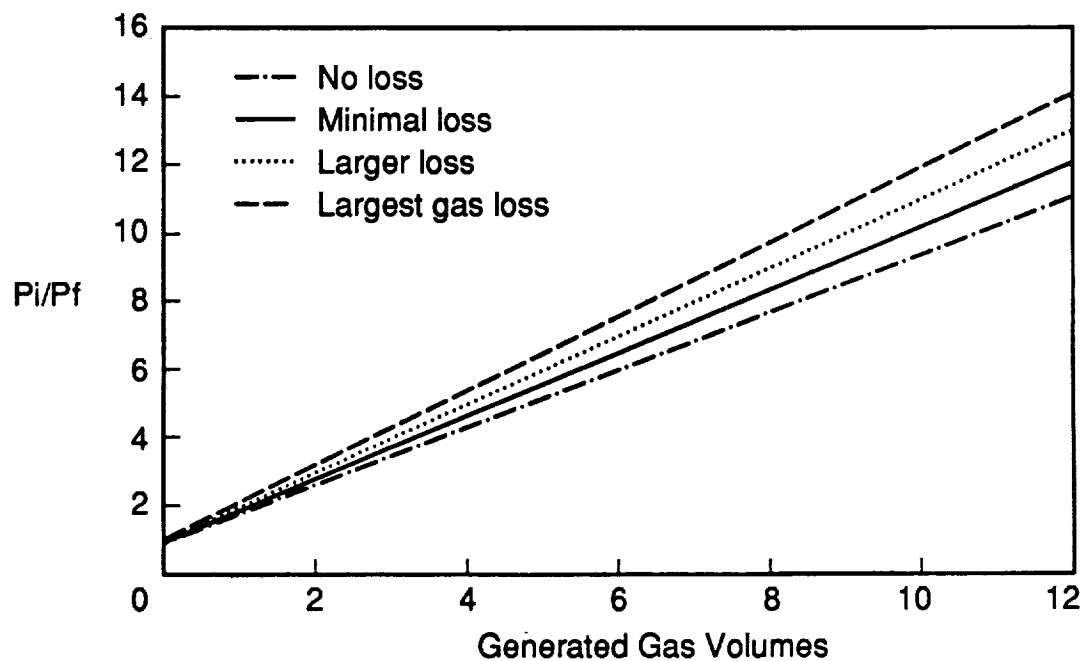
Gas Volumes Created with Decompression Cases with Varying Supersaturation



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Figure A-2

Gas Volumes Created with Decompression Case with Loss of Saturated Liquid

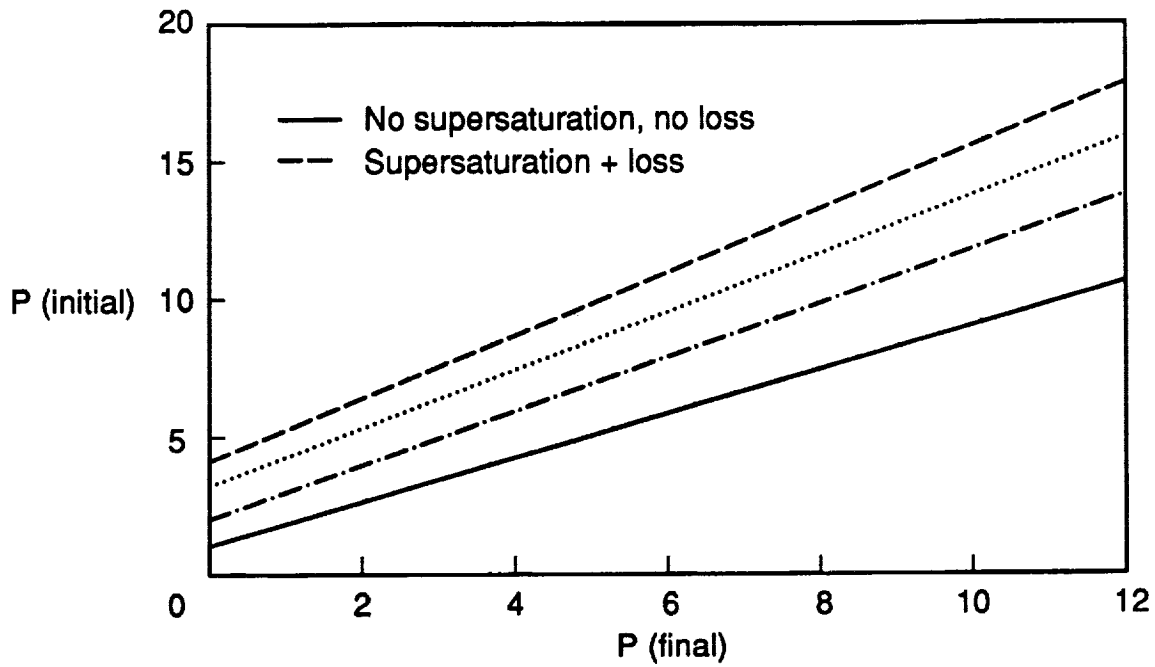


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Figure A-3

Ascent Constraint Plot

Generation of M-Values

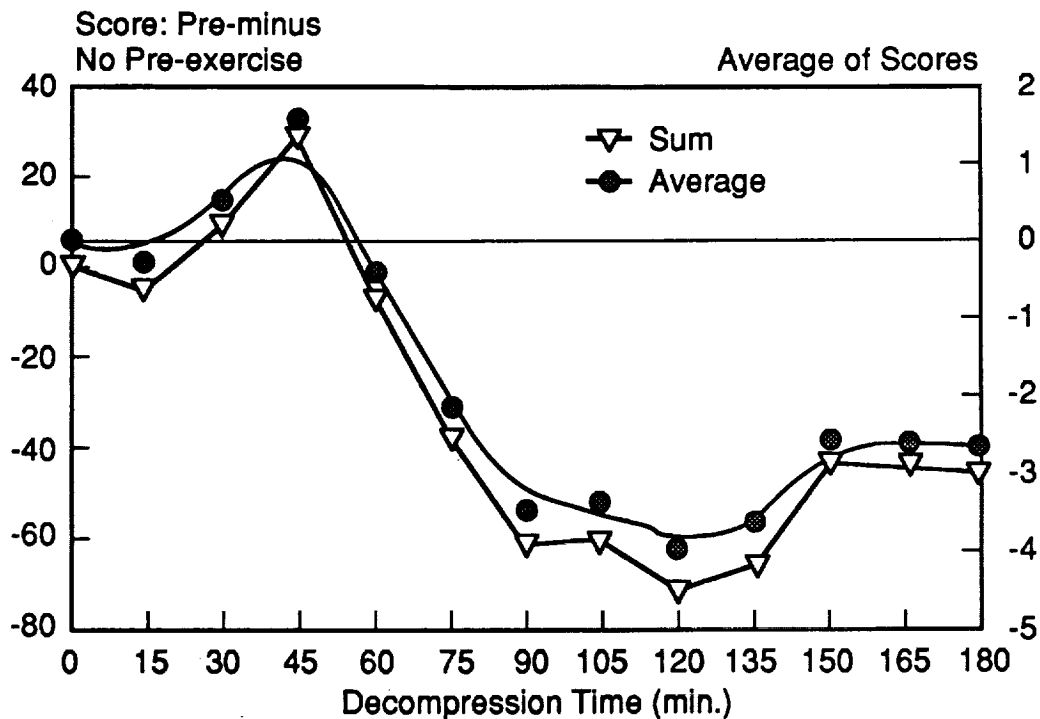


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Figure A-4

Effect of Prior Exercise on Gas Phase Formation

Pre-exercise (+) vs. No Pre-exercise (-)

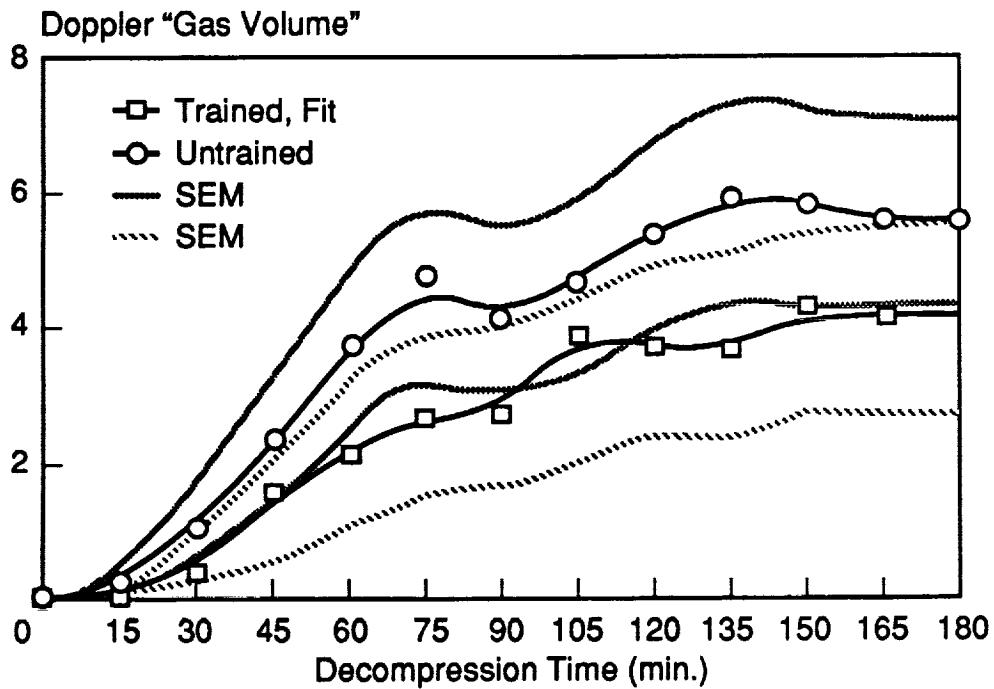


From the data of K.V. Kumar, M.D.
NASA/Johnson Space Center, 1990

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Figure A-5

Doppler "Gas Volume" in Trained and Untrained Individuals



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Figure A-6

APPENDIX B

Example of Usage of the Time-Intensity Method

- A. The Doppler bubble data from a study of two different protocols of pre-decompression exercise is collected in a set manner and recorded as "right arm, right leg, left leg, left arm." The raw Spencer Doppler Grades would be tabulated, for example, (0,2,3,0).
- B. These raw Spencer Grades would be multiplied by factors to convert them to estimated Doppler "gas volumes." These factors are given in table II.
- C. The converted data of the example in paragraph 1 then becomes (0,4,9,0). The sum of these is taken as the "gas volume" for that Doppler monitoring interval.
- D. In a tabular form, we would obtain for an extended experiment of several hours, for example:

<u>Time [min.]</u>	<u>Subject A (Protocol A)</u>	<u>Subject A (Protocol B)</u>
0	0	0
15	0	1
.		
.		
90	15	23.5
etc.		
	<u>sum</u>	<u>sum</u>

E. The time-intensity index for an individual is the sum of all "gas volumes" divided by the number of monitoring sessions. Considerable ease in the analysis procedure is realized by having a standard tabulation method so that all experiments contain the same number of monitoring sessions. Computer programs such as Lotus 1-2-3 can then be instructed to average all "cells" for all monitoring sessions for every subject to produce an average for any one type of exposure (for example, tissue ratio, exercise level, pre-decompression condition, etc.).

F. In one study in which the pre-decompression activity was varied, the individual performed both activities and was compared against him/herself. An analysis of Doppler bubble data by the simple "bubbles detected/not detected" methodology yielded no difference in the protocols. However, a pair-wise analysis gave the results shown in figure A-5. Looking at individuals who had an active life-style (physically trained) *vs.* those with a sedentary one (untrained), we obtain figure A-6.

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